U.S. DEPARTMENT OF COMMERCE
National Technical Information Service

PB-296 903

The Health and Environmental Impacts of Lead and an Assessment of a Need for Limitations

Battelle Columbus Labs, OH

Prepared for

Environmental Protection Agency, Washington, DC Office of Toxic Substances

Apr 79

United States
Environmental Protection
Agency

Office of
Toxic Substances
Washington DC 20460

EPA-560/2-79-001
April 1979

Toxic Substances

EPA

PB 296903

The Health and Environmental Impacts of Lead

An Assessment of the Need for Limitations,

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA-560/2-79-001

2.

3. R PB296903

4. TITLE AND SUBTITLE
The Health and Environmental Impacts of Lead:
Assessment of the Need for Limitations

5. REPORT DATE April, 1979

6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)
Ewing, R.A., Bell, M.A., and Lutz, G.A.

8. PERFORMING ORGANIZATION REPORT NO.

9. PERFORMING ORGANIZATION NAME AND ADDRESS
Battelle Columbus Laboratories
505 King Avenue
Columbus, Ohio 43201

10. PROGRAM ELEMENT NO. 2LS810

11. CONTRACT/GRANT NO. 68-01-4318

12. SPONSORING AGENCY NAME AND ADDRESS
Office of Toxic Substances (TS-792)
Environmental Protection Agency
Washington, DC 20460

13. TYPE OF REPORT AND PERIOD COVERED Final Report

14. SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES

16. ABSTRACT

This report reviews the available literature on lead and its compounds concerning physical and chemical properties, industrial technology and alternatives, environmental sources and emissions, environmental distribution, ecological effects, human health effects, and exposures to lead from various media to determine the need for additional limitations on lead. More than 30 existing regulations directly or indirectly limiting exposure to lead have been identified. In reviewing the need for further limitations, the report addresses two specific questions: (1) What are the present levels of exposure in various population groups, and (2) Do these exposure levels represent a significant health hazard? An attempt is made to identify the relative contributions to total human exposure attributable to air, drinking water, and dietary intake. Using 11 assessment factors, the report prioritizes 12 exposure source classifications which might merit consideration for possible limitations. The impacts on health, the environment, and the economy of these potential limitations remain to be evaluated.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS		b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
lead	alloys	toxicity	
lead oxide	metal	environmental effects	
white lead	batteries	technology	
red lead	shot	regulation	
lead sulfide	pigments		
litharge	gasoline		
tetraethyl lead	solder		

18. DISTRIBUTION STATEMENT Unlimited distribution through The National Technical Information Service Springfield, VA 22151	**19. SECURITY CLASS** *(This Report)* Unclassified	**21. NO. OF PAGES** 516
	20. SECURITY CLASS *(This page)* Unclassified	**22. PRICE** PCA22 / MF-A01

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

1

EPA-560/2-79-001
APRIL, 1979

THE HEALTH AND ENVIRONMENTAL IMPACTS OF LEAD AND AN ASSESSMENT OF A NEED FOR LIMITATIONS

Contract No. 68-01-4318

Project Officer

Elbert L. Dage
Office of Toxic Substances
Washington, D.C. 20460

OFFICE OF TOXIC SUBSTANCES
U.S. Environmental Protection Agency
Washington, D.C. 20460

1-A

DISCLAIMER

This report has been reviewed by the Office of Toxic Substances, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the U.S. Environmental Protection Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

ABSTRACT

This study was performed to assess the health and environmental impacts of lead and its compounds and the need for additional limitations upon its production, use, or disposal. The investigation included an analysis of the lead industry and the uses of lead, estimates of the nature and quantities of lead dispersed to the environment and their effects on the ecosystem and on man, and an analysis of the scope and status of present limitations.

An evaluation of candidates for the application of limitations disclosed that leaded gasoline and canned or processed foods ranked highest. Since the former has already been addressed and new limitations promulgated, attention was focused on lead intake from foods and beverages.

Food may account for from 60 to as much as 90 percent of lead intake for the general population. Lead is naturally present in foods and this fraction of the body burden is probably unavoidable. However, significant lead contamination is inadvertently introduced into processed foods, primarily from the lead solder used in food cans. It has been estimated that as much as two-thirds of the lead in canned foods results from the solder. No other lead limitation considered appeared to offer as great a potential benefit to the entire population as a reduction of lead in canned foods. Limitations directed at the dietary mode of exposure will benefit sensitive segments, e.g. children, and the general population alike.

Analysis of dietary intakes and blood lead concentrations indicated that reductions in mean blood leads of 1μg/dl or more, provide a significant and beneficial reduction in normal blood lead levels in the general population. This level of reduction can be achieved by improvements in food can manufacturing technologies and procedures, while still retaining the economic and technological benefits of soldered can construction.

Considerable progress toward these goals is presently being made by the can industry, in cooperation with the Food and Drug Administration; it is recommended that the results of these efforts be evaluated before further action is taken.

Other potential sources of lead ingestion considered included lead-glazed foodware, leaded glass, decal-decorated glass tumblers, solder-joined food vessels, and pewter ware. On the basis of the evidence available, all of these appeared to be minor sources, not requiring further investigation at this time.

This report was submitted in fulfillment of Contract No. 68-01-4318 by Battelle Memorial Institute under the sponsorship of the U.S. Environmental Protection Agency. This report covers the period September 30, 1976, to April 30, 1979, and work was completed as of May 30, 1979.

THE HEALTH AND ENVIRONMENTAL IMPACTS OF LEAD AND ASSESSMENT OF A NEED FOR LIMITATIONS

TABLE OF CONTENTS

Page

Abstract iii

Tables xii

Figures. xvii

Acknowledgement. xix

1.0 General Summary/Environmental Assessment. 1

2.0 Introduction. 10

3.0 Properties of Lead and Its Compounds. 12

- 3.1 Physical and Chemical Properties 12
  - 3.1.1 Inorganic Compounds of Lead 15
  - 3.1.2 Organic Compounds of Lead 17
  - 3.1.3 Isotopes of Lead. 21

- 3.2 References 23

4.0 The Lead Industry 25

- 4.1 Overview/History 25

- 4.2 Lead Producing Industry. 28
  - 4.2.1 Primary Lead. 28
    - 4.2.1.1 Mining 28
    - 4.2.1.2 Beneficiation. 31
    - 4.2.1.3 Smelting and Refining. 32
    - 4.2.1.4 Production and Products. 34
  - 4.2.2 Secondary Lead. 38
    - 4.2.2.1 Scrap Pretreatment 42
    - 4.2.2.2 Smelting 43
    - 4.2.2.3 Refining 43
  - 4.2.3 Supply/Demand Relationships 45
    - 4.2.3.1 Stockpiling. 48

TABLE OF CONTENTS (Continued)

Page

4.2.3.2 Refined Lead Prices 48
4.2.3.3 Reserves. 51

4.3 Lead Consuming Industries 52

4.3.1 Storage Batteries. 53
4.3.1.1 Manufacture. 53
4.3.1.2 Production and Industry Structure. . . . 60
4.3.1.3 Future Projections. 63
4.3.2 Gasoline Antiknock Additives 64
4.3.2.1 Manufacture. 64
4.3.2.2 Production and Industry Structure 71
4.3.2.3 Future Projections 78

4.3.3 Lead Oxides and Pigments 84
4.3.3.1 Manufacturing. 84
4.3.3.2 Production and Industry Structure 87
4.3.3.3 Future Projections 90
4.3.4 Inorganic Lead Compounds 90
4.3.4.1 Manufacturing 95
4.3.4.2 Production and Industry Structure. 96
4.3.4.3 Future Projections 105
4.3.5 Miscellaneous Uses of Metallic Lead 106
4.3.5.1 Ammunition. 107
4.3.5.2 Solder. 108
4.3.5.3 Sheet Lead. 109
4.3.5.4 Cable Covering. 109
4.3.5.5 Weights and Ballast 110
4.3.5.6 Type Metal. 110
4.3.5.7 Pipes, Traps, and Bands, and Caulking Lead 111
4.3.5.8 Brass and Bronze and Bearing Metals . . . 112
4.3.5.9 Casting metal 113
4.3.5.10 Collapsible Tubes and Foil. 113
4.3.5.11 Miscellaneous Uses 114
4.3.6 Organic Lead Compounds 114

4.4 References 115

5.0 Introduction of Lead Into the Environment. 121

5.1 Summary 121

5.2 Sources. 121

5.2.1 Lead Producing Industry 121
5.2.1.1 Primary Lead 121
5.2.1.2 Secondary Lead 127
5.2.1.3 Secondary Brass and Bronze. 128
5.2.2 Lead Consuming Industry. 129
5.2.2.1 Storage Battery Manufacture 129

TABLE OF CONTENTS (Continued)

Page

5.2.2.2 Gasoline Antiknock Additives 129
5.2.2.3 Lead Oxides and Pigments. 135
5.2.2.4 Inorganic Lead Compounds. 137
5.2.2.5 Miscellaneous Uses of Metallic Lead. . . 138
5.2.3 Indirect Sources of Lead 140
5.2.3.1 Iron and Steel Production 140
5.2.3.2 Cement manufacture 141
5.2.3.3 Combustion of Fossil Fuels. 142
5.2.3.4 Solid Waste Incineration 143
5.2.3.5 Sludge Disposal. 143

5.3 References. 145

6.0 Ecological Effects 151

6.1 Summary. 151

6.2 Effects on Terrestrial and Aquatic Protista. 153

6.2.1 Terrestrial Protista. 154
6.2.2 Aquatic Protista. 155

6.3 Effects on Plants. .156

6.3.1 Metabolism, Uptake, and Absorption. 157
6.3.2 Yields 159
6.3.3 Organolead Compounds. 160

6.4 Effects on Aquatic Biota. 160

6.4.1 Invertebrates. 160
6.4.2 Fishes. 161

6.5 Effects on Terrestrial Biota. 163

6.5.1 Invertebrates. 163
6.5.2 Vertebrates. 163
6.5.2.1 Birds. 163
6.5.2.2 Mammals. 165

6.6 Environmental Interactions. 166

6.6.1 Physical and Chemical Behavior. 166
6.6.2 Environmental Transport and Transfers. 171
6.6.2.1 Air/Plants. 171
6.6.2.2 Soil/Plants. 175
6.6.2.3 Plants/Animals 176
6.6.2.4 Aquatic Systems. 177

TABLE OF CONTENTS (Continued)

Page

6.7 References. 180

7.0 Effects on Humans. 192

7.1 Summary. 197

7.2 Biological Pathways. 197

7.2.1 Uptake and Absorption 197
7.2.1.1 Inhalation. 197
7.2.1.2 Ingestion. 197
7.2.1.3 Skin Absorption. 200
7.2.1.4 Placental Transfer. 200
7.2.2 Transport and Distribution. 202
7.2.3 Elimination. 211

7.3 Toxic Effects. 212

7.3.1 Toxicities of Inorganic Lead Compounds. 212
7.3.2 Reproductive Effects. 215
7.3.2.1 Fertility Reduction. 215
7.3.2.2 Mutagenicity. 217
7.3.2.3 Teratogenicity. 217
7.3.3 Carcinogenicity. 218

7.4 Lead Poisoning in Children. 219

7.4.1 Adverse Effects of Lead. 220
7.4.2 Lead Dose Necessary to Produce Adverse Effects. . . 220
7.4.3 Estimated Lead Intake in a Child with Pica for Paint. 226

7.5 Epidemiology. 229

7.5.1 Studies of General Population. 230
7.5.2 Intermediate Level Studies. 242
7.5.2.1 Mobile Emissions Sources. 243
7.5.2.2 Populations Impacted by Stationary Sources. 245
7.5.3 Studies of Occupational Groups. 252
7.5.3.1 Threshold Levels for Clinical and Subclinical Effects. 252

7.6 Organic Lead. 264

7.6.1 Uptake and Absorption. 266
7.6.2 Tissue Distribution. 267
7.6.3 Elimination. 268
7.6.4 Toxic Effects. 268

TABLE OF CONTENTS (Continued)

Page

7.6.4.1 Acute Toxicity of Various Organolead Compounds. 268
7.6.4.2 Neuropathology. 269
7.6.4.3 Metabolic Effects. 269
7.6.4.4 Enzyme Effects. 271
7.6.4.5 Reproductive Effects. 271
7.6.5 Epidemiologic Studies. 271

7.7 References. 273

8.0 Lead in the Environment. 292

8.1 Summary 292

8.2 Lead in Air. 296

8.2.1 Air Lead Concentrations in the U.S. 297
8.2.2 Localized Source-Oriented Studies. 301
8.2.2.1 Antiknock-Related Studies. 301
8.2.2.2 Smelter-Related Studies. 305

8.2.3 Trends in Atmospheric Lead Concentrations. . . . 309

8.3 Lead in Water. 314

8.3.1 Water Lead Concentrations in the U.S. 316
8.3.2 Lead Concentrations in Drinking Water. 320
8.3.3 Trends in Water Lead Concentrations. 324

8.4 Lead in Soils. 324

8.4.1 Soil Lead Concentrations in the U.S.. 325
8.4.2 Localized Source-Oriented Studies. 325
8.4.3 Lead in Urban Dusts. 328
8.4.4 Trends in Soil Lead Concentrations. 330

8.5 Lead in Foods and Beverages. 331

8.5.1 Determinants of Lead Content. 332
8.5.2 Lead Content of Individual Food Commodities. . . 333
8.5.2.1 Food and Forage Plants. 333
8.5.2.2 Processed Fruit and Vegetable Products. 336
8.5.2.3 Meat, Fish, and Poultry Products. . . . 337
8.5.2.4 Milk and Infant Formulas. 339
8.5.2.5 Beverages, Bakery Products, Sugar, and Condiments. 341
8.5.3 Adventitious Lead in Processed Food. 344

TABLE OF CONTENTS (Continued)

Page

8.6 Source Contributions to Lead Intake by Humans. 351

  8.6.1 Lead Intake From Air. 351
  8.6.2 Lead Intake From Drinking Water 355
  8.6.3 Lead Intake From Soil and Other Non-Food Items. . . 356
  8.6.4 Lead Intake From Foods and Beverages. 357
  8.6.5 Relative Contributions of Various Media to Body Burdens. 364
    8.6.5.1 Adults 364
    8.6.5.2 Children 367
  8.6.6 Recommendations Concerning Safe Levels of Dietary Lead. 373
  8.6.7 Effects of Reducing Adventitious Lead in Canned Foods on Dietary Lead Intake. 376
    8.6.7.1 Methods for Deriving Food Consumption Levels. 376
    8.6.7.2 Daily Dietary Lead Intakes from Different Foods for Various Age-Sex Groups. 384

8.7 Lead Uptake/Blood-Lead Relationships. 390

  8.7.1 Relationship of Blood Lead Levels to Lead Exposure in Humans. 390
  8.7.2 Distributional Characteristics of Blood Lead Values in General Populations. 392
  8.7.3 Effects of Reducing Dietary Intake on Mean Blood Lead Levels. 393
    8.7.3.1 Approach to Estimation . . . .
    8.7.3.2 Example Case 395

8.8 References. 401

9.0 Assessment of the Need for and Possible Approaches to Limitations on Lead 415

9.1 Summary. 415

9.2 Need for Limitations 418

  9.2.1 Acute Lead Poisoning. 418
  9.2.2 Limitations at Low Lead Levels. 419

9.3 Approaches to Limitations. 426

  9.3.1 Criteria for Prioritization 426
  9.3.2 Prioritization of Possible Limitations. 427
    9.3.2.1 Leaded Gasolines 428
    9.3.2.2 Adventitious Lead in Food and Beverages. . . 430

TABLE OF CONTENTS (Cont.)

Page

9.3.2.3 High-Lead Paint in Homes. 431
9.3.2.4 Lead-Based Paints. 432
9.3.2.5 Printing Inks. 433
9.3.2.6 Lead-Acid Storage Batteries. 434
9.3.2.7 Lead in Plastics and Rubber 434
9.3.2.8 Lead Ammunition 435
9.3.2.9 Miscellaneous Uses of Metallic Lead . . . 435
9.3.2.10 Emissions from Lead in Industry Processes. 436
9.3.2.11 Emissions from Indirect Sources. 436

9.3.3 Non-Structural Approaches 437

9.4 Existing and Proposed Limitations. 438

9.4.1 Direct Limitations on Lead 441
9.4.1.1 Nonferrous Ore Mining and Milling. . . . 441
9.4.1.2 Ferroalloy Ore Mining and Milling. . . . 442
9.4.1.3 Primary Lead. 442
9.4.1.4 Primary Copper. 442
9.4.1.5 Electroplating. 442
9.4.1.6 Lead Storage Battery Manufacture. 443
9.4.1.7 Chlorine-Caustic 443
9.4.1.8 Lead Monoxide. 443
9.4.1.9 Chrome Pigments. 443
9.4.1.10 Television Picture Tubes. 443
9.4.1.11 Hand Pressed and Blown Glass. 444
9.4.1.12 Lead-Sheathed Rubber Hose. 444
9.4.1.13 Gasoline Antiknock Additives. 444
9.4.1.14 Lead-Based Paint. 445
9.4.1.15 Lead-Based Paint in Federally-Owned or Assisted Housing. 446
9.4.1.16 Occupational Exposure to Lead. 447
9.4.1.17 Drinking Water Standard. 447
9.4.1.18 Evaporated Milk. 447
9.4.1.19 Ambient Air Standards. 448
9.4.1.20 Lead Arsenate. 448
9.4.1.21 Lead Shot. 449
9.4.1.22 Ocean Dumping 449
9.4.2 Indirect Limitations. 449
9.4.2.1 Primary Lead Smelters. 449
9.4.2.2 Primary Copper Smelters. 450
9.4.2.3 Secondary Lead Smelters. 450
9.4.2.4 Secondary Brass and Bronze Ingot Production Plants. 450
9.4.2.5 Ferroalloy Production Facilities. 450
9.4.2.6 Electric Arc Steel Furnaces. 450
9.4.2.7 Incinerators. 450
9.4.2.8 Sludge Incinerators. 450

TABLE OF CONTENTS (Continued)

Page

9.4.2.9 Portland Cement Plants. 450
9.4.2.10 Petroleum Storage. 450
9.4.2.11 Gasoline Vapor Recovery. 451
9.4.3 Recent Enabling Legislation. 451

9.5 Case Study: Analysis of Alternatives for Limitations on Lead Intake from Foods and Beverages. 452

9.5.1 Lead in Processed Foods and Beverages. 452
9.5.1.1 Food Can Technology. 452
9.5.1.2 Alternatives to Lead-Soldered Food Cans. . 458
9.5.1.3 Good Manufacturing Practices. 460
9.5.1.4 Economic Effects of Limitations Alternatives. 463
9.5.1.5 Industry Trends and Outlook. 471

9.5.2 Lead-Glazed Foodware. 472
9.5.2.1 Glaze Technology. 473
9.5.2.2 Historical Perspective. 475
9.5.2.3 Alternatives to Lead Glazes. 476

9.5.3 Miscellaneous Sources of Ingested Lead. 477
9.5.3.1 Decal-Decorated Tumblers 477
9.5.3.2 Lead Glass 478
9.5.3.3 Solder-Joined Food Vessels. 478
9.5.3.4 Pewter. Ware. 479
9.5.3.5 Drinking Water. 479

9.6 References. 481

Appendix. 484

Lead Intakes from Various Foods for Selected Age-Sex Groups 484

TABLES

Number Page
3.1 Physical Constants for Lead. 13
3.2 Oxidation-Reduction Potentials for Lead. 14
3.3 Solubilities of Inorganic Compounds of Lead. 16
3.4 Properties of Tetraethyllead and Tetramethyllead 20
4.1 Consumption Patterns for Lead in Selected Countries, 1976. . . 27
4.2 Lead Smelters and Refineries 36
4.3 U.S. Production of Refined Lead and Antimonial Lead at Primary Refineries, by Source Material, 1966-1975. 37
4.4 Standard Specifications for Pig Lead (B29-55). 39
4.5 Lead Recovered from Scrap Processed in the United States, 1966-1975 41
4.6 Supply of Lead in the United States, 1967-1977 46
4.7 U.S. Imports of Lead by Types of Material, 1967-1977 47
4.8 U.S. Exports of Lead by Type of Product, 196701977 49
4.9 Selected Compounds of Lead and Their Uses. 54
4.10 U.S. Lead Consumption by Class of Product, 1967-1976 58
4.11 Lead Consumption in the United States, By Products, 1973-1976. 59
4.12 Estimated Structure of Storage Battery Industry. 62
4.13 Lead Alkyl Manufacturers and Estimated Capacities. 65
4.14 Production and Consumption of Lead Alkyls, 1966-1976 73
4.15 Average Lead Content of Gasolines, 1965-1978 74
4.16 Consumption of Gasoline and Lead Anti-Knock, 1955-1978 77
4.17 Gasoline Consumption Forecast. 80
4.18 Effect of Phasedown on Consumption of Lead in Gasoline 83
4.19 Lead Pigments--U.S. Shipments, By Industry, 1971-1975. 89
4.20 Plants Producing Lead Pigments 91
4.21 Inorganic Lead Compounds and Their Uses. 93
4.22 Producers of Lead-Based Plastic and Rubber Stabilizer-Type Materials. 98
4.23 Estimated Consumption of Lead-Based Stabilizers in Plastics and Rubber Industries, 1974. 99
4.24 Producers of Lead-Based Paint Driers 101
4.25 Production of Selected Lead-Based Paint Driers 102
4.26 Producers of Miscellaneous Lead-Based Chemicals Produced by More than One Company 103
4.27 Miscellaneous Lead-Based Chemicals Produced by Only One Company. 104
5.1 Estimated Releases of Lead to the Environment. 127
6.1 Concentrations of Lead Reported Toxic or Lethal to Fish. . . . 162
6.2 Lead in the Above-Ground Portion of Grasses as a Function of Distance from Traffic or Industries. 172
6.3 Lead in Crops, Greenhouse Studies. 173
6.4 Lead in Crops, Highway Studies 173

TABLES (Continued)

Number Page

6.5 Mean Concentration of Lead in Tissue of Small Mammals from Roadside and Control Sites 177
7.1 Lead Ingestion and Excretion of a Normal Human Subject 199
7.2 ALA-D Activity in Liver and Distribution of Lead in Various Organs in Rats 201
7.3 Tissue Lead Concentration in Children and Adolescents. 203
7.4 Variation in Human Tissue Lead Concentration with Geographical Locations. 207
7.5 Tissues Ranked on the Basis of Overall Mean Concentrations of Lead 209
7.6 Age-Related Increases of Lead Concentration in Body Tissues. . 210
7.7 Representative Toxicities and Occupational Standards for Seven Commercially Important Inorganic Lead Compounds. 213
7.8 Calculated Lead Intake and Absorbed Dose from Paint Pica . . . 227
7.9 Calculated Daily External Dose and Associated Internal Dose. . 228
7.10 Blood Lead Levels of Selected Human Populations. 231
7.11 Mean Air Lead Exposure for the Five Groups Examined by Azar, et al. (1975). 237
7.12 The Impact of Air Lead on Blood-Lead Levels; A Comparison of Actual Data to Predictions Using Mathematical Model. 241
7.13 Descriptions of Lead Plants Investigated, 1975-1976. 255
7.14 Blood-Lead Levels in Lead Plant Workers. 257
7.15 Percentage of Workers with Symptoms of Lead Poisoning by Blood Lead and Erythrocyte Protoporphyrin Level at Three Lead Plants 257
7.16 Frequency of Symptoms Among 22 Cases of Lead Poisoning, Utah, 1976 259
7.17 Frequency of Symptoms in Lead Smelting Plant Employees, Minnesota, 1976. 259
7.18 Cutaneous Absorption of Lead Compounds 267
7.19 Representative Toxicities and Occupational Standards for Tetraethyl and Tetramethyl Lead. 270
8.1 Lead Concentrations in Surface Air From Selected Sites Along the 80th Meridian, 1967. 298
8.2 Concentration of Lead in the Atmosphere of Three Cities, 1961-1962 300
8.3 National Average Ambient Atmospheric Lead Concentrations: Quarterly Composites 302
8.4 List of 31 Urban Areas with Quarterly Lead Concentrations Equalling or Exceeding 1.5 μg/m^3 in 1975 303
8.5 Effect of Proximity to Highway in an Urban Environment on Air and Blood Lead Concentrations. 306
8.6 Lead Content of Various Marine Waters. 314
8.7 Regional Summary of Lead in Surface Water of the U.S 317
8.8 Number of Samples of Surface Waters Exceeding Specified Concentrations of Lead 319
8.9 Lead Content of Tap Water From Two Surveys of Distributed Water. 322

TABLES (Continued)

Number Page

8.10 Lead Content of Roadside Soil and Grass as a Function of Distance From Traffic and Soil Depth. 326
8.11 Lead in Dirt in Detroit. 329
8.12 Lead Content of Cereals and Vegetables 334
8.13 Lead Content of Fruits 336
8.14 Lead Content of Processed Fruits and Vegetable Products. . . . 337
8.15 Lead Content of Meat, Fish and Poultry Products. 338
8.16 Lead Content of Milk and Infant Formulas 340
8.17 Lead Content of Beverages. 342
8.18 Lead Content of Bread and Baked Products 343
8.19 Lead Content of Sugar, Spices, Condiments, and Miscellaneous Foods. 343
8.20 Variations in Lead Concentrations with Time in Selected Canned Food Products. 348
8.21 Potential Daily Intake of Lead by Inhalation 352
8.22 Deposition of Lead Inhaled by Man. 353
8.23 Estimated Dietary Intake of Lead by Food Class 359
8.24 Rank Order of Lead Findings by Food Class. 360
8.25 The Effects of Various Assumptions of the Value of Trace and Non-Detections on Estimates of Dietary Intake. 361
8.26 Lead Content of Adult Foods Sampled by FDA Heavy Metals in Foods Survey; FY 1974. 363
8.27 Lead Content of Baby Foods Sampled by FDA Heavy Metals in Foods Survey; FY 1974. 364
8.28 The Relative Contributions of Various Media to Lead Intake and Absorption: Adult Males. 366
8.29 Estimated Total Absorbed Dose at Varying Air Lead Concentrations: Young Adult Males. 369
8.30 The Relative Contribution of Various Media to Lead Intake and Absorption in 3-Year Old Children. 371
8.31 Total Daily Lead Absorbed By 3-Year Non-Pica Child and Percentage Attributable to Various Media under Varying Environmental Conditions 374
8.32 Estimated Lead Content of Various Foods. 377
8.33 Food Consumption in Grams Per Day for Various Age-Sex Groups. 380
8.34 Daily Dietary Intake of Lead for Various Age-Sex Groups and The Effect of Reducting Lead in Canned Foods by One-Half or Two-Thirds 385
8.35 Reduction in Daily Lead Intake for Children from Various Food Classes after Elimination of 1/2 or 2/3 of the Lead in Canned Foods. 386
8.36 Reduction in Daily Lead Intake for Adults from Various Food Classes after Elimination of 1/2 or 2/3 of the Lead in Canned Foods. 387
8.37 Geometric Mean Air- and Blood-Lead Values for 11 Study Populations. 391
8.38 Frequency Distribution of Blood Lead Values in Two Hypothetical Populations of Adult Females. 398

TABLES (Cont.)

Number Page

9.1 Blood-Lead Levels versus Lowest-Observed-Effect Levels. . . . 422
9.2 Assessment of Factors Characterizing a Need and Feasibility of Limitations on Lead. 429
9.3 Summary of Existing and Proposed Federal Regulations Governing Limitations on Lead. 439
9.4 Comparison of Results of 1974 and 1976 Canned Food Surveys. 462
9.5 Lead Content of Infant Juices Packed in Metal Containers. 464
9.6 Major U.S. Merchant Producers of Metal Cans, 1975. . . 465
9.7 Summary of Estimated Costs of Alternative Food Can Manufacturing Techniques. 470

TABLES

APPENDIX A

Table A-1. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males and Females Under 1-year Old. A-1

A-2. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males and Females 1-2 Years Old. A-2

A-3. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males and Females 3-5 Years Old. A-3

A-4. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males and Females 6-8 Years Old. A-4

TABLES

APPENDIX A (Cont.)

A-5. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males 15-17 Years Old. A-5

A-6. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Females 15-17 Years Old. A-6

A-7. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males 20-34 Years Old. A-7

A-8. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Females 20-34 Years Old. A-8

A-9. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Males 55-64 Years Old. A-9

A-10. Daily Intake of Lead From Various Foods and the Effect of Reducing Lead Content of Canned Foods: Females 55-64 Years Old. A-10

FIGURES

Number | Page

3.1 Solubility and species distribution for Pb(II) in soft water. 18
3.2 Solubility and species distribution for Pb(II) in hard water. 19
4.1 Simplified flow sheet for production of primary lead. . . . 29
4.2 Geographic locations of domestic primary lead smelters and refineries. 35
4.3 Lead scrap flow. 40
4.4 Secondary lead smelting process. 44
4.5 Trends in the lead industry in the United States. 50
4.6 Simplified flow sheet for storage battery manufacture. . . . 61
4.7 Sodium-lead alloy process for the manufacture of tetraethyl lead. 66
4.8 Electrolysis process for the manufacture of tetramethyl lead. 69
4.9 Nationwide trends in gasoline lead content, 1965-1985. . . . 75
4.10 Passenger car use of premium gasoline. 76
4.11 Projected consumption of grades of gasoline, 1970-1990. . . 81
4.12 Estimated lead content of leaded and total gasoline pool, 1970-1985. 81
4.13 Simplified flow sheet of processes for the manufacture of lead-based white pigments. 86
4.14 Simplified flow sheet of processes for manufacture of lead chromate-based pigments. 88
6.1 Ecologic flow chart for lead showing possible cycling pathways and compartments. 167
7.1 Levels of blood lead in volunteers exposed to 10.9 μg/m^3 lead sesquioxide. 205
7.2 Dose-response relationship for the effects of internal doses of lead ad Pb-B on erythrocyte protoporphyrin . . . 221
7.3 Mean blood lead concentration for epidemilogic and experimental respiratory exposures. 232
7.4 Blood lead levels and corresponding mean air lead levels. . 234
7.5 Blood lead versus total air lead. 239
7.6 Hypothetical distribution curves of blood lead concentrations of a population exposed first to 200 μg Pb/m^3 and then to 500 μg/m^3. 253
8.1 Air lead values as a function of traffic volume and distance from the highway. 304
8.2 Concentrations of metals in air as a function of distance from the El Paso smelter. 308
8.3 Concentration of particulate lead in air. 310
8.4 Relative abundance of lead in airborne particulates and in soil samples. 310
8.5 Average urban ambient air lead concentrations vs. lead anti-knock consumption, with phasedown. 312
8.6 Depth profiles of common lead concentration in the central northeast Pacific. 315
8.7 Annual mean level of lead in U.S. streams, 1975. 318
8.8 Percentage of Boston tap water samples exceeding lead standard. 323

FIGURES (Cont.)

Number Page

8.9 Mean lead concentrations of Boston tap water samples by type of sample. 323
8.10 Distribution of lead in canned and bottled products. . . 345
8.11 Lead distribution in single cans of tomato paste. 345
8.12 Lead distribution in single cans of tomato paste after storage at room temperature. 347
8.13 Effect of seam length/volume ratio on the lead level of can contents. 347
8.14 Lead absorbed by man. 365
8.15 The relative contribution of various media to total absorbed lead dose. 368
8.16 Daily dietary intake of males, by age. 388
8.17 Daily dietary intake of females, by age. 389
8.18 Predicted distribution of blood lead levels at two different mean blood leads for adult women, 20-34 years old. 397

ACKNOWLEDGEMENTS

Other Battelle-Columbus staff members contributed specialized expertise in the preparation of this document, including Mr. John B. Hallowell in the area of nonferrous metals, Mr. James L. Otis on alkyl leads, Mrs. J. Caroline Warling and Mr. David A. Savitz on the compilation and analysis of diets and lead intakes, and Mr. Raymond W. Hale on general industry economics.

We also wish to express our appreciation for the cooperation received from EPA staff of The Office of Toxic Substances, particulary the project officer, Elbert L. Dage, who provided support throughout the program.

1.0 GENERAL SUMMARY

This general summary is intended to serve as an overview of the investigation and highlight the principal findings; more detailed summaries are included within the individual chapters.

Analysis of the findings of this investigation of the health and environmental effects of lead supports the conclusions of prior investigators examining this problem, and leads to a number of general conclusions on the need for additional controls on lead, and suggested ways and means for applying these limitations most advantageously.

Lead is already a highly regulated substance, and most of the exposure hazards leading to acute lead poisoning have already been closely regulated. These are primarily occupational exposures, to which the population in general is not exposed. Less intense, but more pervasive sources of lead intake, which may affect the general population, such as lead in gasoline antiknock additives, have also been addressed in recent years, and the combination of the gasoline lead phasedown and the ambient air quality standard for lead has placed this major environmental source of lead finally under control.

Of the ten remaining candidates for consideration of limitations, ingested lead, specifically lead in foods, was identified as the preeminent source of lead intake for the general population, and was selected for further investigation.

Most of the lead intake from food is attributed to lead accidentally introduced during the canning process. Methods for achieving large reductions in this adventitious lead, short of the total elimination of lead-soldered food cans, are available, and the can-making industry is vigorously pursuing this approach, in cooperation with the Food and Drug Administration. The reductions in mean blood lead concentration of the general population which can be achieved by these evolutionary improvements are calculated to be so significant that they should not be overlooked. The ongoing efforts of the FDA in this area are proving so effective that it is recommended that the results of this program be awaited and evaluated before further action is taken.

Lead is a dense, soft, malleable, easily fused, non-corroding metal, almost as durable in the environment as a noble metal. As described in Chapter 3, most lead compounds are moderately to highly insoluble. This characteristic has an important bearing on the lack of mobility of lead in soil and in water, and partially explains why lead has not posed a large problem in ecosystems, in spite of the fact that man has over the years discarded and dispersed millions of tons of lead to the biosphere.

Lead is also a rare metal. Its average abundance in the earths crust has been estimated to be only about 16 ppm. Nevertheless, there are a surprising number of commercially exploitable deposits, and lead is a commercially important base metal. Lead is durable and relatively easy to refine or rerefine. Thus, secondary lead is recovered from scrap and recycled, on a scale comparable to that of primary lead (See Chapter 4).

The United States is the leading producer of primary lead, producing approximately 500,000 to 600,000 metric tons per year, mostly from deposits in southeastern Missouri. About the same quantity of secondary lead is produced each year.

Consumption of lead in the U.S. is inextricably related to the automobile industry. In recent years, from 800,000 to 1,000,000 metric tons (from 60 to 70 percent of total lead consumption) have gone to the manufacture of storage batteries and gasoline antiknock additives. Lead storage batteries represent the only large segment of the lead industry with a foreseeable significant growth potential. Except for a few relatively minor uses, all other uses of lead are relatively stagnant or declining. Much of this decline is directly related to increasing pressures to find substitutes for or to minimize the use of this toxic metal. Lead-containing paints and paint driers are one example of this trend; whereas lead-based house paints were formerly very widely used, no house paint manufactured today may contain in excess of 0.06 percent lead, i.e. must be lead-free.

Lead's uses vary from those that are totally dissipative to those almost non-dissipative; as described in Chapter 5, most of the introduction of lead to the environment arises directly from dissipative uses. Alkyllead gasoline antiknock additives is the premier example of a dissipative use which has accounted for almost all of the lead dissipated initially to the air, and ultimately to soil and water. On the other hand, lead in storage batteries, the largest single use (consuming typically about 750,000 tons/yr of lead) is an essentially non-dissipative use. The lead in batteries is not consumed in service, and an estimated 80 percent is recycled. Even that which escapes recovery and recycle is in such a compact and immobile form that its dispersion and transfer through the ecosystem is minimal.

Lead is also introduced to the environment to some extent by indirect routes not associated at all with the use of lead or lead compounds, for example, from the combustion of coal or the firing of cement calcine. Other indirect sources include iron and steel production and the incineration of solid wastes or sewage sludges. Fortunately, as discussed in Chapter 5, the quantities of lead emitted from these indirect sources are small in comparison to the major sources of lead emissions. Part of the reason for this is that required control of particulate emissions concomitantly also provides control of particulate lead. The contribution from these sources to the body burden of the general population is small, estimated to possibly be from a fraction of a percent to 1-2 percent at the worst.

The ecological effects of lead (Chapter 6) tend to be unspectacular and subtle. Although excessive levels of lead in the environment have the same kinds of effects upon terrestial and aquatic systems as most other toxic substances, such excessive levels are the exception. Most such incidents appear to have been associated with large-scale emissions of lead from industrial point sources. The massive amounts of lead which have been released to the environment from the combustion of leaded gasolines have not elicited propartionally serious ecological effects, possibly because the dispersion is so uniform, and environmental concentrations are below the level at which effects are apparent. Although lead has been shown to bioconcentrate in some fish species, there is no evidence of biomagnification. The poisoning of waterfowl from eating lead shot has prompted regulatory action to substitute iron shot in the principal U.S. flyways to reduce this hazard. Laboratory and field studies have shown that lead inhibits plant growth and produces other adverse biochemical phenomena, although such effects are minimal at usual ambient concentrations. Ecologically speaking, the indications are that lead is a nonessential element that exhibits a low degree of potential toxicity to plants and a high degree of potential toxicity to animals.

The major toxic effects of lead have been upon people (Chapter 7). Lead poisoning has been recognized as an occupational problem for centuries; and many restrictions have been placed upon conditions in the work place to reduce this hazard. Within the past year the Occupational Safety and Health Administration promulgated regulations reducing by one-half the allowable workroom air lead concentration, from 100 to 50 $\mu g/m^3$; and this had a few years ago been halved from the earlier 200 $\mu g/m^3$ level. This category of exposure to lead can be characterized as one of very high occupational risk to a small group of people, which is being addressed by OSHA, but is not an environmental risk to which the general population is exposed.

Lead's toxic effects range from acute lead poisoning (seen in some occupationally exposed individuals and in children who ingest large quantities of lead by eating paint chips) about which there is general agreement on its manifestations and epidemiology, to the other extreme, found at threshold exposures, where there is considerable scientific controversy about the effects. There is still controversy as to the maximum level of lead exposure which produces no adverse effects, arising in part from the subtle nature of these threshold effects.

Attempts to establish a dose-response relationship between environmental lead concentrations and adverse health effects and/or the relative contribution of each of the various sources of exposure to body burden have met with only limited success. The underlying difficulty in such an analysis may be traced to the multiplicity of sources of lead exposure and the difficulty in accurately quantifying individual exposure.

Although it suffers from some drawbacks, blood lead, expressed as micrograms of lead per 100 milliliters of blood (μg/dl), is generally accepted as the best available analytical measure of recent lead exposure and readily available body lead. Also for the purposes of this study, the effects criteria proposed by EPA in presenting ambient air quality criteria for lead were adopted. At the same time it was recognized that these estimated no-effect levels are based on limited populations, and there is no evidence that they apply to the population in general.

Lead may be absorbed into the body by inhalation, ingestion, skin absorption, or placental transfer. Various studies indicate that 37-50 per cent of inhaled lead is deposited in the lungs. In adults, approximately 7.5-10 percent of ingested lead is absorbed in the gastrointestinal tract; recent data indicate that this is not the case with children, in children absorption is much higher, 44-50 percent. These recent results for children suggest that earlier estimates of acceptable daily intake for young children may have been grossly overestimated, and points up the desirability of inclusion of adequate safety margins in establishing allowable intakes, and in considering possible limitations on lead.

The critical effect of inorganic lead compounds is interference with heme synthesis. Short-term inhalation of organic lead compounds primarily affects the central nervous system. However, organic lead poisoning is fundamentally a wholly occupational risk, to which the general population is not normally exposed, and was, therefore, not considered further in this study.

Lead appears also to have an effect on reproductive ability; and may have teratogenic effects in man, although the evidence for this is not definitive. Evidence to date does not indicate that lead is carcinogenic for humans.

Lead poisoning in children is a serious but preventable problem. The primary source of this is ingestion of paint chips containing lead and to a lesser degree, ingestion of dust and dirt contaminated by weathering of leaded paints, automotive emissions, and in some limited areas, smelter emissions. Regulation of lead levels in paint, together with phasedown of leaded gas and ambient air quality standards should be expected to reduce future risks effectively. Much leaded paint is still present in older housing. This source of exposure will remain significant until pre-World War II housing is replaced with newer dwellings.

Prevention of lead poisoning in children residing in these older homes is primarily a matter of parental education (especially regarding good housekeeping to minimize the amount of paint chips, dust, etc. available to the young child) early detection and follow-up of lead poisoned children to detect and eliminate the specific source(s) of exposure from the child's environment. Unless the source is eliminated or controlled following diagnosis and treatment, recurrance of lead poisoning is probable.

One source of lead intake for the general population is air. The consensus of studies of diverse general populations suggests that the contribution of ambient air lead to blood lead is in the range of 0.6-2.0 μg/dl per 1 μg/m^3 in air, although the correlation of air lead and blood lead is not high over the narrow ranges of lead concentration found in ambient air. The correlation is more definitive at high air leads with indications that the rate of change in blood lead decreases with increasing air lead.

Lead is found in all three media, air, water, and soil, although most attention in recent years has been focused on lead in air. As discussed in Chapter 7, lead in the atmosphere is a potentially significant contributor to intake and body burdens, from the annual combustion of 150,000 tons of lead in gasoline antiknock additives. As the consumption of lead antiknock has begun to decrease, from the peak of 220,000 metric tons, achieved in 1970, improvements in air lead concentrations are beginning to become apparent. The annual average for urban stations in the National Air Sampling Networks (NASN) decreased from 1.23 μg/m^3 in 1971 to 0.89 μg/m^3 in 1975. On the basis of the relationship derived between gasoline antiknock consumption and annual average air lead concentration, it is estimated that the annual average for 1978 was about 0.58 μg/m^3, and following the leaded gasoline phasedown, will decline to about 0.3 μg/m^3 by 1980 and thereafter. When air lead concentrations attain this level, inhalation will become a rather minor contributor of lead to man's body burden.

Lead concentrations in U.S. surface waters are essentially all below 50 ppb (μg/l), about the solubility of lead in hard water. The available data suggest that the median concentration for U.S. drinking water supplies is about 10 ppb, and 80 percent of public water supplies are below 20 ppb lead. Thus, it is not surprising that the contribution of drinking water to the lead burden of the general population is small. A few localized trouble spots exist in New England and the Pacific Northwest, where lead water pipe was formerly used in domestic water systems, and where the water is soft and has a low pH.

The mean soil lead concentration in the U.S. is approximately 16 ppm, and is changing only slowly. The lead in soils is relatively unavailable to crops, and does not appear to pose a health risk to the general population. Urban dust and dirt is another matter. Lead concentrations in dust and dirt in and around dwellings as high as 1,000 to 11,000 ppm have been found; the latter levels represent extreme cases. Adults are essentially unaffected by high dust and dirt lead concentrations, but these high concentrations represent a real health risk to inner city children living in deteriorated housing, because of the hand-to-mouth tendencies of small children.

It is generally accepted that the major source of lead for the average person is food. Neither air nor water compare with food as a source of lead; depending on the specific contributions of these other sources, food may represent from 60 to as much as 90 percent of lead intake. All foods naturally contain lead in varying concentrations, though for many foods this may be only a few hundreths ppm. Fresh milk, an important food for infants and small children, will contain 0.05 ppm or less, for example. This part of man's daily intake of lead is probably unavoidable, but also probably not harmful. Most of the adventitious lead in the average diet arises from canned foods, and is derived from the lead solder seam of the can. It is generally accepted that this is a very substantial fraction, as much as one-half to two-thirds of the total lead content of the foods, as eaten. Some of this lead is dissolved by the food, especially the more acid and agressive foods, but the evidence indicates that a probably more important source is particulate lead (spatter, dust, powder) which lodges inside the can during the soldering process.

Adventitious lead in food is controllable, either by abandonment of the three-piece lead-soldered can (which would totally eliminate the problem) or by improving can-making technology and quality control. The latter option has much to recommend it.

Using the latest publicly-available FDA and Department of Agriculture dietary data, and estimates of lead contents of foods, both fresh and packed, drawn from the literature, lead intakes were calculated for a number of age-sex groups. By similar calculations, it was possible to estimate the reductions in lead intake accruing if one-half or two-thirds of the lead in canned food could be eliminated by modifications in can-making technology.

Data were available on the mean blood lead level of one special group (20 - 34 year-old females) which permitted the calculation of the population distribution curve as a function of blood lead level with and without the projected reduction. These calculations indicated that a reduction of 1.3 μg/dl in blood lead would result for this group if two-thirds of the lead in canned food were eliminated, which would reduce by about half the number of women in this age group with blood lead levels (above the standard cut-off point of 25 μg/dl) in the range where evidence of hematological changes begin to appear. Similar benefits can be projected for other population segments, using the same technique.

Even after levels of lead in the environment are determined, the routes by which this lead enters man are identified, and the contributions of the various media estimated. the question still remains as to whether there exists a need for additional limitations on lead. The answer to this is not immediately self-evident since lead is already controlled by a multiplicity of existing or imminent Federal regulations. More than 30 regulations limiting exposure to lead either directly or indirectly were identified (Section 9). Addressing the need for additional limitations on lead, the answer to this question depends in part on the answer to the two other questions: (1) What is the present level of exposure in various segments of the population, and (2) Does sustained exposure at these levels represent a significant health hazard?

An unequivocal answer is not possible to either of these questions and therein lies some of the problem surrounding lead. Presently there are no data concerning blood lead levels of representative national samples, although as an approximation the evidence indicates 15-20 μg/dl as a reasonable estimate for adults, and perhaps 20-25 μg/dl for children. The answer to the second question is even more uncertain, depending as it does on the consensus evaluation of the significance of the subtle biochemical effects of lead at low doses, and whether or not these are reversible, and what their long term effect is, especially on children. As has been made evident in the discussions concerning the establishment of an ambient air lead standard, this is a question surrounded by a considerable scientific controversy.

Nevertheless, upon examination of the broadly-stated problem the conclusion was reached that the question that whether or not a "threshold" blood lead level at which effects could have long-term significance overlapes the existing blood lead concentration in the populace did not need to be answered. Even if there is no overlap, the gap between the two does not provide the safety factor needed, and prudence dictates that this safety margin be increased. Acceptance of this premise leads naturally to the conclusion that additional limitations of some sort on lead warrant consideration.

Analysis of eleven candidates for limitations were made to establish priorities for their further consideration. Although the needed data were not available to permit quantifying possible benefits, as in the case of the 20-34 year-old females with respect to lead reductions in ingested food, a comparative evaluation was possible. Adventitious lead in food and leaded gasolines ranked well above other candidates.

Analysis of the limitations already enacted to control lead in gasoline and projections of their effects on lead emissions, led to the conclusion that there is no pressing need for additional limitations in this area at this time, with a recommendation that the results of these regulations be awaited and evaluated before further action is taken.

As noted above, analysis of the relative lead intakes from inhalation and ingestion demonstrated conclusively that the ingestion of food and water was the most predominant source of average daily lead intake for the general population unless air lead concentrations reached abnormally high levels. Additionally, in considering specific limitations, one should concentrate first on those producing the maximum benefit to the largest group of persons or the most sensitive segments of the population, or ideally, to both. Limitations directed toward the dietary mode of exposure would benefit sensitive segments and the general population alike.

An exception to the generalization that food constitutes the maximum source of lead intake is, of course, the child with pica who eats lead-containing paint chips, plaster or dirt. Ingestion of relatively minute quantities of such materials can far outweigh all other sources of exposure for these children and all too often has led to clinical lead poisoning (Section 7.4). However, a mode of application of a limitation on lead to eliminate this very important source of lead was not identified. Increased emphasis on parental education programs and early detection and treatment appears to offer the most effective solution to this problem at this time.

Thus, for the general population, reduction of lead accidentally introduced into food appeared to be the limitation candidate offering the greatest benefit to the largest number of people, in addition to especially benefiting the sensitive population segment of young children.

There are alternatives to the standard three-piece can with a soldered side seam, and billions of cans are made without a soldered seam. Steel three-piece cans may be made with welded or cemented side seams; or two-piece cans with no side seam at all can be formed by the drawing and redrawing or drawing and ironing processes. Billions of two-piece aluminum cans are manufactured for beer containers. However, none of these alternatives are well suited to the rigors of aseptic canning, nor to the economic manufacture of the wide variety of sizes of cans needed by the food industry.

However, significant reductions in adventitious lead in foods packed in three-piece soldered cans have been shown to be possible, and the can industry has already embarked on an evolutionary program in cooperation with the Food and Drug Administration, to accomplish this, still based on the three-piece soldered can. This program involves some modifications of the can line solder station, to prevent the entrance or to clean up the lead solder spatter and dust from the interior of the can, since it has been shown that this was the primary contributor to lead pickup, rather than dissolution of the structural lead in the soldered seam.

An important element in this improvement program is the Good Manufacturing Procedures (GMP) concept utilized by FDA in its monitoring efforts over the manufacture of foods and drugs, which includes strong emphasis on quality control. The effectiveness of this evolutionary approach was demonstrated in an industry test in 1976 on a number of can making lines which had been modified to reduce accidental lead pickup. Lead contents of thirteen foods (selected as those likely to be fed to young children) averaged 0.19 ppm, a 41 percent reduction from the 0.32 ppm average of the comparable 1974 survey. When the entire sanitary food can industry has been similarly modified and GMP applied, a 40 to 50 percent reduction in lead content in all canned food can be anticipated, with a significant benefit to mean blood lead values.

Capital costs to incorporate these can making line modifications and cleanup improvements into the approximately 1,085 lines in the U.S. producing sanitary food cans was estimated by the Can Manufacturers Institute at about $45,000 per line, or nearly $50 million for the entire industry. Added operating costs (investment amortization only) were estimated to be a nominal $0.10 - 0.30/M cans.

Capital costs for the alternatives of adding an organic polymer stripe over the seam, or a 360-degree spray over the entire interior were $70,000 and $660,000-930,000 per line, respectively. Added operating costs were in the $0.20 - 0.75/M can range. All of the lead-free alternative forming methods were much more costly, ranging from $1 to 3 million in new capital cost per line, and from $3-6/M can added operating costs.

Limitations on the lead content in foods would have to take into account the varying and unknown lead content of the original raw food, which pretty well rules out the approach of specifying a maximum lead content of processed foods. The GMP approach, already utilized by FDA for similar product quality control problems, appears to be a more flexible and adaptable control mechanism, which should accomplish the desired objective, and is recommended, at least as an interim measure. If this approach is successful, the much more costly solution of total elimination of lead from food cans may not be necessary.

Other potential sources of ingested lead considered for possible limitation included: lead glazed foodware; lead glass; decal-decorated glass tumblers, solder-joined food vessels; and pewter. Although quantitative data on these potential sources of lead intake were sparse, it was clear that they contribute very little to the body burden of the general population, and they were accorded a low priority for more detailed investigation. No further investigation of these minor sources of ingested lead is recommended at this time.

2.0 INTRODUCTION

Lead is an ubiquitous element in the biosphere, not surprising for a metal which has been used by man since antiquity. Therefore, it will be present in the human body, with the body burden depending on the level of environmental exposure. It is clear that lead levels in the environment have been gradually increasing ever since the beginning of the industrial revolution; and the rate of increase accelerated when gasoline antiknock additives came on the scene. Hopefully, the controls now being applied to this foremost dissipative use of lead will reverse this trend; there is some evidence that this has begun to happen.

The U.S. Environmental Protection Agency has been charged by the U.S. Congress with the general responsibility of protecting man's environment. The Agency has also been charged with the implementation and enforcement of numerous specific acts aimed at this goal. Some of the major acts include The Clean Air Act, The Federal Water Pollution Control Act, The Resources Conservation and Recovery Act., The Safe Drinking Water Act, and The Toxic Substances Control Act. Under the latter act, EPA has the responsibility of controlling and regulating substances posing an "unreasonable risk or injury to health or the environment."

Lead is a known toxic substance and the toxic properties of lead have been recognized for centuries, along with its special properties which have been beneficially utilized by man. Which, if any, of the manifold uses of lead and its compounds pose an "unreasonable risk" was a matter requiring further definition. There have been numerous studies of various aspects of the health and safety hazards posed by some specific uses of lead, e.g., in paint, but there do not appear to have been any studies examining the uses and releases of lead on an overall basis, relative to performing an assessment of the need for and nature of possible limitations.

Such an overall assessment was the objective of this investigation. A further, more specific objective, was to assess the present and future need for limitations, and if a need were indicated, to identify the types of limitations which appear to be the most justified and most effective.

This study, then, evaluates the nature and structure of the lead industry, the myriad uses of lead and its compounds, the losses of lead to the environment and their fate, the resultant level of health and environmental hazards, and where the most effective reduction of those hazard levels might be effected.

Lead is so ubiquitous in the environment that a portion of man's intake is essentially irreducible. Thus, efforts to minimize this intake necessarily focus on the reducible fraction.

As the investigation proceeded it became evident that while all foods and beverages naturally contain lead in trace concentrations, the sources of this lead are so widespread and diffuse as to be almost uncontrollable. Thus, significant reductions in the lead content of unprocessed food appeared unlikely. On the other hand, a substantial fraction of the lead intake from food of the general population was determined to be adventitious, added accidentally in the processing and packing of food, and significant reductions in this portion of man's lead intake appeared to be attainable.

Since the resources available to this study permitted investigation of but a few limited avenues of approach to possible limitations on lead, lead intake via ingestion was selected as the limitation candidate of highest priority, focusing primarily on the reducible fraction associated with canned food.

3.0 PROPERTIES OF LEAD AND ITS COMPOUNDS

3.1 PHYSICAL AND CHEMICAL PROPERTIES

Lead is a soft metal with a bluish-gray color when freshly cut. Its bright luster soon disappears upon exposure to air because of the formation of lead oxide. The important physical constants of lead are presented in Table 3.1. It is assigned atomic number 82 in the periodic classification of the elements, and has several naturally occurring isotopes which result in an average atomic weight of 207.19. Because lead has four electrons in its outer shell, it falls in Group IV of the periodic table, and consequently, might be expected to show a normal valence of +4 in its compounds. However, the 2 "s" electrons are difficult to ionize and as a result the usual valence of lead in ionic compounds is +2 rather than +4. In acid solutions lead is a fair reducing agent; in alkaline solutions it is a rather strong reductant. The more important oxidation-reduction potentials for lead and its compounds are presented in Table 3.2 (Latimer, 1952).

Lead has good resistance to neutral solutions, here the oxide and carbonate are the corrosion products, and is fairly resistant to alkaline solutions. Lead is resistant to sulfuric, sulfurous, phosphoric and chromic acids, but it is moderately attacked by hydrochloric and hydrofluoric acids. It is strongly corroded by nitric, acetic and formic acids and moderately affected by organic chlorides and aqueous solutions of metal chlorides. In the presence of oxygen most organic acids react with lead to form the corresponding organolead salts (Hamner, 1974).

Lead's high density (11.3 grams per cubic centimeter), its softness, and relatively low melting point enable easy forming and casting. These physical characteristics account for the numerous uses of lead (see Section 4.3). Lead forms low-melting alloys with tin, arsenic, antimony, bismuth, cadmium, and calcium, singularly and collectively. These alloys are fabricable by commercial processes (Hack, 1967). Alloys are also made from secondary lead which is recovered from scrap. Lead does not readily alloy with zinc, iron, nickel, and other high-melting metals. Alloys of lead can be extruded, drawn, rolled, cast, stamped, spun, and applied as a coating to other metals by hot dipping, electroplating, or spraying (Lead Industries Association, 1952). Thick layers of lead can be bonded to steel and other metals.

Table 3.1 PHYSICAL CONSTANTS FOR LEAD[a]

Melting point, C	347.43
Boiling point, C	1740
Specific gravity, at 20 C	11.3437
at 327.43 C	10.686
at 650.0 C	10.302
at 850.0 C	10.078
Vapor pressure, at 987 C, mm Hg	1.0
at 1167 C	10.0
at 1417 C	100.0
at 1508 C	200.0
at 1611 C	400.0
Surface tension, at 350 C, dyn/cm^2	442
at 400 C	438
at 500 C	431
Viscosity, at 441 C, cp	2.116
at 551 C	1.700
at 703 C	1.349
at 844 C	1.185
Specific heat, at 0 C, cal/g	0.0297
at 20 C	0.0306
at 100 C	0.0320
at 327 C	0.0390
at 500 C	0.037
Latent heat of fusion, cal/g	5.86
Latent heat of vaporization, cal/g	203
Thermal conductivity, at 20 C, cal/(cm^2) (C/cm) (sec)	0.083
at 100 C	27.021
at 327.43 C	94.6
at 600 C	107.2
at 800 C	116.4
Magnetic susceptibility, 10^{-6} egs units	0.12
Brinell hardness (cast)	4.2
Element bond length, Pb-Pb- at 25 C, Å	3.5003
Electrical resistivity, micro-ohms/cc at 20 C	20.648
Refractive index, sodium light	2.01
Velocity of sound, cm/sec x 10^5	1.227
Tensile strength, lb/sq in at 20 C	1920
Linear coefficient of expansion, per C x 10^{-5}	2.93

[a] Adapted from McKay. In: Encyclopedia of Chemical Technology, Vol. 12, A. Standen (ed.), Used by permission of Interscience Publishers, New York. (c) John Wiley and Sons, Inc., 1967.

Table 3.2 OXIDATION-REDUCTION POTENTIALS FOR LEAD[a]

Reaction	$\Delta E°$, volts
$Pb \rightarrow Pb^{+2} + 2e$	+ 0.126
$Pb^{+2} \rightarrow Pb^{+4} + 2e$	- 1.7
$Pb + SO_4^{-2} \rightarrow PbSO_4 + 2e$	+ 0.356
$Pb + 2OH^- \rightarrow PbO + H_2O + 2e$	+ 0.58
$Pb^{+2} + 2H_2O \rightarrow PbO_2 + 4H^+ + 2e$	- 1.455
$PbSO_4 + 2H_2O \rightarrow PbO_2 + 4H^+ + SO_4^{-2} + 2e$	- 1.685
$PbO + 2OH^- \rightarrow PbO_2 + H_2O + 2e$	- 0.248
$Pb + 2Cl^- \rightarrow PbCl_2 + 2e$	+ 0.268
$Pb + 2Br^- \rightarrow PbBr_2 + 2e$	+ 0.280
$Pb + 2I^- \rightarrow PbI_2 + 2e$	+ 0.365
$Pb + S^{-2} \rightarrow PbS + 2e$	+ 0.980

[a] Adapted from Latimer, The Oxidation State of the Elements and Their Potentials in Aqueous Solutions, 2nd Edition, (c) 1952, pp 151-155. Reprinted by permission of Prentice-Hall, Inc., Englewood Cliffs, New Jersey.

3.1.1 Inorganic Compounds of Lead

The divalent lead compounds resemble those of the alkaline earth elements. The sulfate, nitrate, and carbonate compounds of lead are isomorphic with the corresponding barium and strontium compounds. Many lead compounds have two or more crystalline forms. The common inorganic compounds of lead (nitrate, chlorate, and acetate) are water-soluble; the chloride is sparingly soluble; and the sulfate, carbonate, chromate, phosphate, and sulfide are insoluble.

Lead monoxide (PbO), one of the most important industrial lead compounds, exists in two forms. At ordinary temperatures, the monoxide exists as reddish-yellow, tetragonal crystals commonly referred to as litharge. At 480 C this transforms into yellow orthorhombic crystals known as massicot. Neither form of the oxide is appreciably soluble in water. Litharge disolves only to the extent of 0.0017 gram/100 ml of water at 20 C, and massicot, 0.0023 gram/100 ml. The melting point of PbO is 888 C; it has appreciable volatility below this. PbO reacts readily with all common acids to form the corresponding salts and dissolves in alkali, though with more difficulty. Hence, PbO serves as a starting point for the preparation of many lead compounds (Thompson, 1967). The major uses of the inorganic and organic compounds of lead are discussed in Section 4.3.

Red lead, Pb_3O_4, contains both divalent and tetravalent lead atoms in a complex crystalline structure. It is prepared by the oxidation of PbO in air at 500 C.

Lead dioxide, PbO_2, is a dark brown solid in which the lead atoms are tetravalent. It is a vigorous oxidizing agent when heated (290 C). PbO_2 can be produced in the laboratory by the anodic oxidation of solutions of lead compounds. Commercially, it is produced by the treatment of an alkaline slurry of red lead with chlorine (Klug and Brasted, 1958).

Values for the solubilities of common inorganic compounds of lead are presented in Table 3.3 (Lange, 1967; Weast, 1974). Lead sulfide has a very low solubility (approximately 0.8 mg/l) and its formation can serve as a convenient method for the removal of lead from aqueous systems. Its solubility is higher than might be expected on consideration of the low solubility product ($Ksp = 7 \times 10^{-29}$). This is due mainly to the hydrolysis of the sulfide ion. (Kolthoff, et al., 1964).

The equilibrium model for the chemistry of aqueous lead developed by Stumm and Morgan (1970) indicates that at low pH and low pe (reducing conditions) elemental lead can be readily dissolved. This has health implications, where soft water with little or no buffering capacity is at a low pH and lead piping is used for potable water. Excessive lead concentrations in such water have been observed in Great Britain and in certain locations in the United States (see Section 8.3).

Table 3.3 SOLUBILITIES OF INORGANIC COMPOUNDS OF LEAD[a,b]

Compound		Water Solubility, g/100 ml	Temperature, C
Name	Formula		
Acetate	$Pb(OOCCH_3)_2 \cdot 3H_2O$	44.3	20
Bromide	$PbBr_2$	0.844	20
Carbonate	$PbCO_3$	1.1×10^{-4}	20
Chlorate	$Pb(ClO_3)_2 \cdot H_2O$	151.3	18
Chloride	$PbCl_2$	0.99	20
Chromate	$PbCrO_4$	5.8×10^{-6}	20
Fluoride	PbF_2	0.064	20
Hydroxide	$Pb(OH)_2$	0.0155	20
Iodide	PbI_2	0.063	20
Nitrate	$Pb(NO_3)_2$	56.5	20
Oxalate	$Pb(OOC)_2$	1.6×10^{-4}	18
Oxide, mono-	PbO	1.7×10^{-3}	20
Oxide, tetra-	Pb_3O_4	5.3×10^{-5}	20
Phosphate	$Pb_3(PO_4)_2$	1.4×10^{-5}	20
Sulfate	$PbSO_4$	4.25×10^{-3}	25
Sulfide	PbS	8.6×10^{-5}	20

[a] Reprinted with permission from Handbook of Chemistry, 10th edition, N.S. Lange (ed.) (c) McGraw-Hill Book Company, 1967.

[b] Reprinted with permission from Handbook of Chemistry and Physics, 55th Edition, R.D. Weast (ed.). (c) CRC Press, Inc., 1974.

In the absence of suspended solids, the solubility of lead in water is dependent on pH, salt content and partial CO_2 pressure. Equilibrium calculations by Davies and Everhart (1973) indicate the solubility of lead to be about 30 ppb in hard water and 500 ppb in soft water. As illustrated by Figure 3.1, below a pH of about 5.4, $PbSO_4$ is the species limiting solubility. Above pH 5.4, $PbCO_3$ and $Pb_2(OH)_2CO_3$ are the limiting factors in lead concentrations in solution. In hard water a pH greater than 6.0 is the level at which the above carbonates are present and cause a corresponding limitation (Figure 3.2). The most important factor in determining lead solubilities is the carbonate concentration which in turn depends on the partial pressure of CO_2 and the pH. The envelope boundary line C_{Pb} shows concentrations of lead in solution. The minimum occurs at a pH just above 9 in soft water and just below pH 9 in hard water.

3.1.2 Organic Compounds of Lead

Organolead compounds are those in which the lead atom is bound directly to one or more carbon atoms. Since Löwig's first synthesis of an organolead compound in 1853, organolead chemistry has developed into one of the largest areas of organometallic chemistry. It has been estimated that about 1200 organolead compounds were known in 1965 (Shapiro and Frey, 1968). Only a few of these achieved commercial status.

The tetraorganolead compounds in which a tetravalent lead atom is bonded to four organic groups through carbon represent the most stable class of compounds. The four organic groups may be alkyl, aryl, cycloalkyl, arylalkyl, thienyl, furyl, and substituted derivatives thereof. The most important of these compounds are the fuel additives, tetraethyllead (TEL) and tetramethyllead (TML).

In general, the alkyl derivatives of lead are highly toxic compounds, and are readily absorbed through the skin (see Section 7.6). TEL and TML are clear, colorless liquids, volatile, nonpolar, nonionic, and soluble in many organic solvents such as hydrocarbons, chloroform, ether, and absolute ethanol. They have very low water solubilities, and are relatively unreactive in air, water or alkaline solutions (Shapiro and Frey, 1968). They are, however, light-sensitive and undergo photochemical decomposition when they reach the atmosphere; because they are readily broken down by light and heat, their presence in the atmosphere is transient (National Academy of Sciences, 1972).

At 50 millimeters of mercury, TEL boils at 108.4 C, and TML at 33.2 C. TML decomposes on heating at 265 C, and TEL at 100 C (Shapiro and Frey, 1968). Some of the other physical properties, as summarized by Gerarde (1964), are presented in Table 3.4.

These compounds decompose readily during the combustion process, thus freeing the radicals to prevent the extremely rapid burning of gasoline. The so-called "regular" gasoline contains 3 to 4 milliliters of the tetralead compounds per gallon. This concentration represents from about 1.8 to 2.5 grams of lead per gallon. To scavenge the lead from the engine,

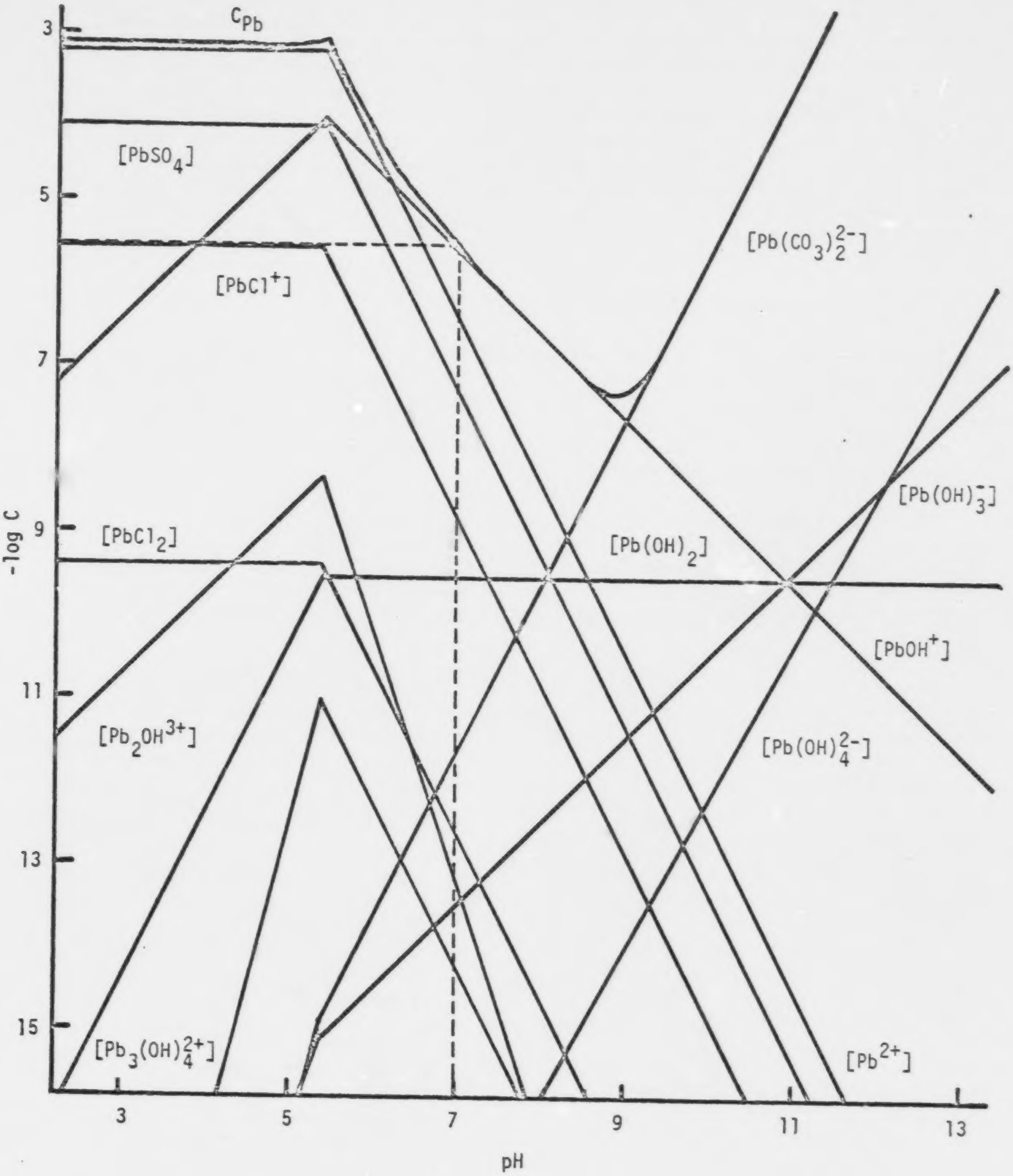


Figure 3.1 Solubility and species distribution for Pb (II) in soft water Source: Davies and Everhart (1973).

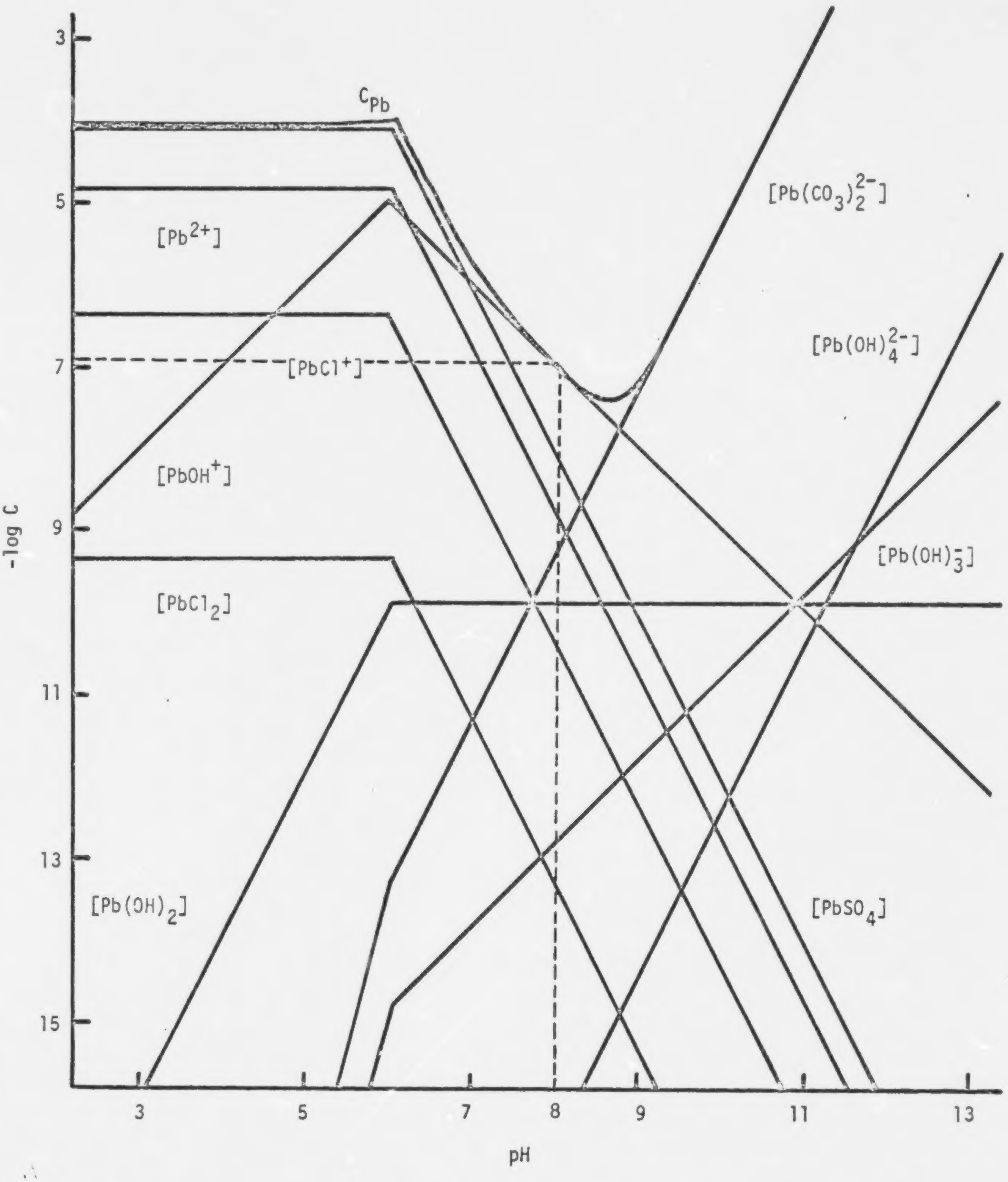


Figure 3.2 Solubility and species distribution for Pb (II) in hard water Source: Davies and Everhart (1973).

Table 3.4 PROPERTIES OF TETRAETHYLLEAD AND TETRAMETHYLLEAD[a]

Physical Properties	Tetraethyllead	Tetramethyllead
Physical form	Watery white oily liquid	Watery white oily liquid
Chemical formula	$(C_2H_5)_4Pb$	$(CH_3)_4Pb$
Odor	Faint, fruity	Faint, fruity (probably odorless in chem. pure state)
Saturated liquid density at 20 C	1.65 g/ml	1.99 g/ml
Vapor pressure at 20 C	0.27 mm Hg	22.5 mm Hg
Boiling point	199 C	110 C
Freezing point	130.2 C	30.3 C
Flash point (open cup)	85 C	About 38 C
Viscosity at 20 C	0.87 cps	0.53 cps
Refractive index nD^{20}	1.520	1.512
Solubility in water at 22 C	0.18 ppm	18.0 ppm
Solubility in gasoline	Soluble in all proportions	Soluble in all proportions

[a] Source: Gerarde. Reprinted with permission from Annual Reviews Pharmacology. (c) Annual Reviews, Inc., 1964.

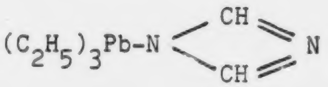
standard antiknock fluids also contain ethylene dibromide and/or ethylene dichloride. The chief lead emission products are (in particles of equivalent diameter between 2 and 10 micrometers) the alpha and beta forms of ammonium chloride and lead bromochloride ($NH_4Cl \cdot 2PbCl\ Br$, $2NH_4Cl \cdot PbCl \cdot Br$), minor quantities of lead sulfate ($PbSO_4$) and the mixed oxide and halide. ($PbO \cdot PbCl \cdot Br \cdot H_2O$) (National Academy of Sciences, 1972). These combustion products, present as solids, are the largest source of atmospheric lead pollution (see Section 5.2.2).

In addition to the tetralead derivatives, other types of organolead compounds are of environmental significance, for example, hexamethyldilead, which consists of six organic groups surrounding a pair of lead atoms:

$$(CH_3)_3Pb\text{-}Pb(CH_3)_3.$$

Lead can also be bonded to organic groups through an oxygen atom giving rise to a variety of alkoxides, such as triemethyllead methoxide, $(CH_3)_3PbOCH_3$. Peroxide structures, an example being tert-butyl trimethyllead peroxide $(CH_3)_3PbOOC(CH_3)_3$ are also known, (Shapiro and Frey, 1968).

Organolead compounds are also formed with Group VI-A elements. A large number of organolead compounds also are known in which the lead is bonded to sulfur, for example, $(CH_3)_3PbSCH_3$. A few compounds have been prepared in which the sulfur is replaced by selenium or tellurium. Organolead compounds in which the lead is bonded to nitrogen are types in which the nitrogen is part of a heterocyclic ring:



(Shapiro and Frey, 1968)

There are, additionally, a number of lead salts of organic acids, such as lead naphthenate, lead di-2-ethyl hexanoate, etc., used in the paint and plastic industries which are, by industry convention, classified as inorganic compounds.

3.1.3 Isotopes of Lead

There are four natural stable isotopes of lead, with the following abundances (Lederer, et al., 1967):

Isotope	Percent
Pb-204	1.4
Pb-206	25.1
Pb-207	21.3
Pb-208	52.7

Pb-204 has no radioactive progenitor. Pb-206, Pb-207, and Pb-208 are produced by the radioactive decay of uranium-238, uranium-235, and thorium-232, with half-lives of 4.5, 0.7, and 14 billion years, respectively (Lederer, et al., 1967). There are four radioactive isotopes of lead occurring as members of these decay series. Pb-211, Pb-212, and Pb-214 have short half-lives, 36.1 min., 10.64 hr., and 26.8 min., respectively. Pb-210 (Radium-D), the longest lived, however, has a half-life of about 20 years, sufficiently long to be useful in environmental studies.

The stable isotope composition of naturally occurring lead ores is known to vary, depending on its geological evolution, and this can be used to determine the geological age of lead deposits. Because of these differences, in some instances isotopic composition may be used, with the assistance of mass spectrometry, to determine the sources of lead contamination. However, because lead is so durable and so much of it is recycled, the pool of recycled scrap, and products derived from it (e.g., TEL) tend to average toward a fairly uniform composition. While this complicates the tracing of the origin(s) of environmental lead by isotopic ratio measurements, the technique has still proven useful.

3.2 REFERENCES

Davies, P. H., and W. H. Everhart. 1973. Effects of Chemical Variation in Aquatic Environments. Vol. III. Lead Toxicity of Rainbow Trout and Testing Application Factor Concept. EPA-R3-73-011c. Office of Research and Monitoring, U.S. Environmental Protection Agency, Washington, D.C. 80 pp.

Hack, C. H., 1967, Lead Alloys. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol 12, A. Standen (ed.) Interscience Publishers, John Wiley and Sons, Inc., New York, New York. pp 248-266.

Hamner, N. E. (compiler). 1974. Corrosion Data Survey. Metals Section, 5th Ed., National Association Corrosion Engineers, Houston, Texas. 283 pp.

Klug, H. P. and R. C. Brasted (eds.). 1958. Comprehensive Inorganic Chemistry, Vol. 7 D. Van Nostrand Co., Inc., Princeton, New Jersey. 302 pp.

Kolthoff, I. M., P. Elving, and E. B. Sandell, 1964, Treatise on Analytical Chemistry, Part II, Vol. 6, Interscience Publishers, John Wiley and Sons, Inc., New York, New York. pp 69-171.

Lange, N. A. (ed.) 1967), Handbook of Chemistry 10th ed., McGraw-Hill Book Company, New York, New York. pp 276-280.

Latimer, W. M. 1952. Oxidation Potentials. Prentice-Hall, Inc., Englewood Cliffs, N.J. pp 151-155.

Lead Industries Association. 1952. Lead in Modern Industry. New York, New York. 230 pp.

Lederer, C. M., J. M. Hollander, and I. Perlman. 1967. Table of Isotopes. 6th Ed., John Wiley and Sons, Inc., New York, New York. 594 pp.

McKay, J. E. 1967. Lead. In: Encyclopedia of Chemical Technology. Vol. 12, A. Standen (ed.), Interscience Publishers, John Wiley and Sons, Inc. New York, New York. pp 207-247.

National Academy of Sciences, Committee on Biologic Effects of Atmospheric Pollutants. 1972. Biologic Effects of Atmospheric Pollutants. Lead. Airborne Lead in Perspective. Washington, D.C. 341 pp.

Shapiro, H., and F. W. Frey. 1968. The Organic Compounds of Lead. Inter-Science Publishers, John Wiley and Sons, Inc., New York, New York. 486 pp.

Stumm, W., and J. J. Morgan. 1970. Aquatic Chemistry. An Introduction Emphasizing Chemical Equilibria in Natural Waters. Interscience Publishers, John Wiley and Sons, Inc., New York, New York. 583 pp.

Weast, R. C. (ed.). 1974. Handbook of Chemistry and Physics. CRC Press, Cleveland, Ohio. 2278 pp. B-100-B-103.

4.0 THE LEAD INDUSTRY

4.1 OVERVIEW - HISTORY

As stated by Callaway (1962), "The discovery of lead is lost in antiquity. The earliest known writings are rich in references to lead. They emphasize its prominence among the metals used by civilized man."

Physical evidence supports the contention that the Egyptians in the 7000-5000 B.C. period used lead, as well as copper, silver, and gold. Also in the pre-Christian era, the Chinese may have circulated flatened lead pieces as coins, while fabled lead deposits were worked by the Phoenicians in Spain, Sardinia, and Cyprus and later by the Carthaginians in Greece. By the time of the Roman empire, the metallurgy of lead was well enough known that a large share of Roman wealth was supplied by silver extracted from the lead ores of Sardinia, Spain, and Britain.

The ancients used lead for coinage and ornamentation. As noted above, the Romans refined lead to recover the silver. Additionally, recent excavations of Roman ruins reveal that they also fabricated the refined lead into pipes and spigots for their water distribution systems, which after 2000 years remain functional. However, the big impetus for lead consumption was the introduction into Europe of gunpowder during the 14th century and the subsequent development of the rifle that fired a malleable metal pellet--a lead shot. Otherwise, lead's corrosion resistance was utilized in roofing for cathedrals and protective encasement of underground pillars.

As western civilization struggled out of the dark ages, Northern European nonferrous metals deposits were discovered and exploited between 700 and 1200 A.D. This was followed by widespread resumption of mining throughout Europe starting about 1400 A.D. Four hundred years of exploitation depleted the high-grade European deposits and by the start of the 19th century European geologists and mining companies were turning to overseas colonies for base metals, including lead.

In the New World, the first authenticated lead mine was established by English settlers in 1721 at Falling Creek, Virginia, to supply metal primarily for bullets and shot. By 1650, the industry was established in New England while concurrently Jesuit missionaries worked some outcrop deposits in Arizona. Expansions of the eastern industry supported the exploration and settlement of territories west of the Alleghenies until

1763 when the southeastern Missouri deposits were opened up on a permanent basis. Then, to protect this burgeoning industry, the First Congress of the United States enacted legislation in 1789 imposing a tariff on lead imports.

After the Civil War and the completion of the transcontinental railway in 1869, mining in Colorado, Utah, Nevada, and eastern California expanded rapidly in response to industrial growth in the northeastern United States. Between 1876 and 1880, the United States surpassed both Germany and Spain as miners and refiners of lead, and has continued to lead in the output of refined lead to the present. With respect to domestic mine production, the United States remained in first place until 1957, when Australia captured the lead, and fell to third place in 1958 behind Australia and the USSR. The opening of the "new lead belt" in Missouri in 1967 revived mining in the United States, which since then has produced at an annual level of 450,000 to 550,000 metric tons (500,000 to 600,000 short tons). In 1975, the United States was the leading country in producing lead from mine sources, accounting for 16 percent of the world total (U.S. Bureau of Mines, 1976). The United States, Russia, Australia, and Canada are the top producers at both the mine and smelter level. These four countries have accounted for 46 percent of world smelter output in the last 10 years.

Modern usage of lead and its alloys probably followed by a short period of time the development of economical methods for producing iron and steel in the 18th and early 19th centuries. Through the late 19th and early 20th centuries, metallic uses of lead dominated the market -- sheet lead for construction and corrosion-resistant chemical equipment and for cable coverings, lead shot for ammunition, lead-tin alloys for pewter-type metal, solders, and bronze (copper-tin) alloys. Red and white lead and chromate pigmments were the principal non-metallic applications. Automotive batteries (during the 1920's) and tetraethyl lead (during the 1930's) emerged as major new uses in the post World War I era. Radiation shielding appears to be the principal new application in the post World War II period although sound attenuation and vibration dampening are expected to have near-term promise.

Consumption patterns for lead vary from country to country because of degree of industrialization, availability of alternative materials, housing customs, and personal transportation modes. Table 4.1 presents apparent consumption patterns in 1976 for the United States, the big four of Europe (France, West Germany, Italy, United Kingdom), and Japan, as reported by the World Bureau of Metal Statistics (1977). The importance of personal automotive transportation in the United States and Japan is readily apparent from the storage battery category. The differences in pigments and chemicals and alloys is less striking. Use for ammunition is either negligible in the other countries, or it is categorized under miscellaneous uses. Europe and Japan still use lead for cable sheathing to a larger extent than in the United States where polymeric sheathings have been preferred. Similarly, constructional applications (pipe, traps, bends), foil applications (collapsible tubes), and corrosion resistant equipment utilize lead in Europe and Japan. In the United States more use is made of copper and plastic pipe, aluminum foil, and stainless steel, rubber

TABLE 4.1. CONSUMPTION PATTERNS FOR LEAD IN SELECTED COUNTRIES, 1976[a]

	Thousands of Metric Tons or Percent					
	United States		European Big Four[c]		Japan	
End-Use Designation[b]	Quantity	Percent	Quantity	Percent	Quantity	Percent
Storage batteries	613	47.2	392	34.1	93	40.4
Cable sheathing	14	1.1	145	12.6	16	7.0
Pigments and chemicals	303	23.4	294	25.6	62	27.0
Alloys	75	5.8	50	4.4	15	6.5
Ammunition	66	5.1	--[d]	--[d]	--[d]	--[d]
Other soft lead products and miscellaneous	226	17.4	267	23.3	44	19.1
Total	1297	100.0	1148	100.0	230	100

[a] Source: World Metal Statistics. Used by permission of World Bureau of Metal Statistics (C)(1977).

[b] End-use designations not specifically defined but believed to be roughly comparable.

[c] France, West Gernmany, Italy, and United Kingdom.

[d] Not reported.

linings, glass linings, or asphaltic compounds, for these uses. Still, the United States consumes nearly one-fourth of all the refined lead used annually.

Depending on the stage of processing, lead ranks third, fourth, or fifth among the industrial metals of the United States. It is third in terms of recoverable metal in domestically mined ores, behind iron ore and copper. It is fourth in terms of primary metal production, behind iron, aluminum, and copper. It is fifth in terms of domestic consumption, behind steel, aluminum, copper, and zinc.

In the following sections, the elements comprising the lead producing and lead consuming segments of the lead industry are described and discussed, including technology, uses, production, geographic locations, and future projections. Although many of the important sources of pollutant emissions are identified in the course of the technology descriptions, their detailed consideration is deferred to the following chapter.

4.2 LEAD PRODUCING INDUSTRY

The producing segment of the lead industry breaks down into two distinct parts, primary lead and secondary lead. The primary lead segment includes the original recovery of lead from ores and concentrates. Since lead is a durable metal, one that is relatively easy to refine or rerefine, there is, accordingly, a major secondary market, comparable in size to the primary metal market. A large fraction of annual U.S. lead consumption conists of recycled secondary lead.

4.2.1 Primary Lead

The primary lead segment is considered to include the mining, beneficiating, smelting, and refining of lead ores and concentrates. The principal operations involved are shown schematically in Figure 4.1.

4.2.1.1 Mining-

In terms of occurrence in the earth's crust, lead is a rare metal. Its relative abundance has been estimated at 16 parts per million (ppm), in contrast to 80 ppm for copper, 125 ppm for zinc, 4800 ppm for iron, or 8000 ppm for aluminum. In spite of this statistical disparity, commercially exploitable deposits of lead-bearing ores occur with remarkable frequency and are widely distributed. Geologically, lead is most frequently associated with zinc and subordinate quantities of silver in replacement deposits in sedimentary rocks. Fissure-vein deposits may have a similar composition but usually are far more complex, including iron and copper as additional major constituents with arsenic, antimony, and bismuth as minor impurities.

Three general types of lead-containing ore are mined in the United States, essentially all from underground mines; they are -- lead ores, lead-zinc ores, and complex ores. Lead ores contain lead as the predominant

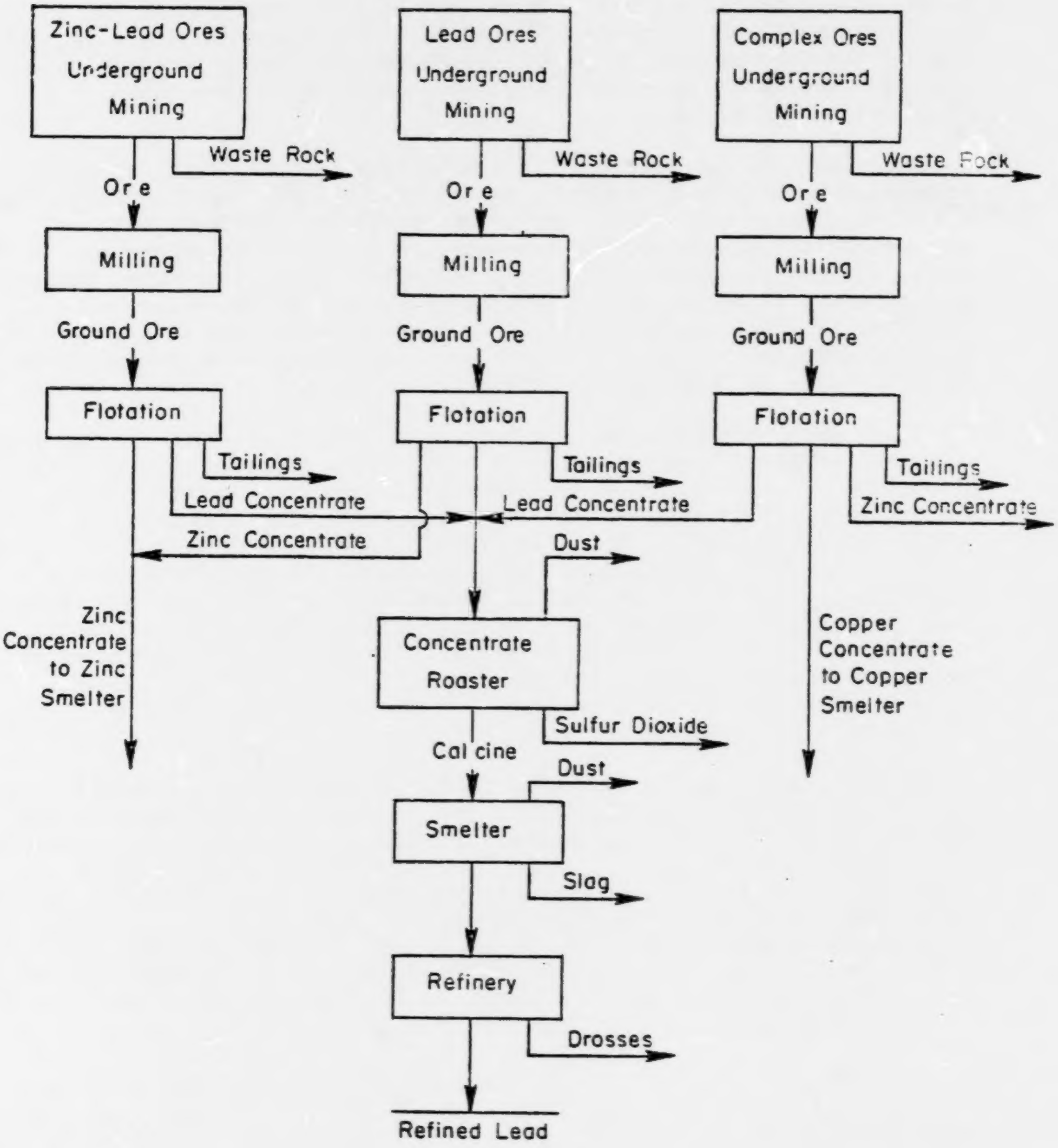


Figure 4.1. Simplified flow sheet for production of primary lead.
Source: Battelle - Columbus

nonferrous metal present and for which the concentrates are treated. Recoverable quantities of other metals may or may not be isolated from waste streams at appropriate stages of processing, depending on economics. Lead-zinc ores have readily recoverable quantities of both lead and zinc and are treated, usually at the beneficiating stage, for segregation of each component. Complex ores contain at least three primary nonferrous metals in quantities and ratios such that economic segregation of individual metals is possible, frequently at the beneficiating stage. The bulk of the complex ores in the United States and Canada contain lead, zinc, and copper as principal constituents plus up to four other recoverable by-products.

Most lead ore deposits contain the sulfide, galena (PbS), commonly associated with sphalerite (ZnS), pyrite (FeS_2), chalcopyrite ($CuFeS_2$) and other sulfides or sulfasalts, any of which may be recovered to yield by-products or coproducts. The near-surface part of some galena ore bodies may be altered to cerussite ($PbCO_3$), anglesite ($PbSO_4$), or other oxidized lead minerals. Mineralization may include marcasite, tetrahedrite, dolomite, calcite, quartz, barite and minerals containing antimony, bismuth, gold and silver. Not all of these materials occur in any given deposit. Lead, as lead, in ore ranges from 1 percent to over 7 percent.

There are about 50 mines in the United States that could recover ores with a significant lead content. The number that actually operate in any given year depends on general economic conditions, world lead prices, and the availability of miners. Under favorable marketing conditions in 1973, the 36 operating mines of that year expanded to 49 in 1974, but fell to 33 in 1975 as markets shrank. Obviously, the variation is attributable, primarily, to the small marginal operations that produce only when supply tends to lag behind demand. The domestic mining industry in 1977 was comprised of about 50 mines in 14 states. Twenty-five mines produced 99 percent of the 1977 output, and the leading 8 mines, all in Missouri, yielded 80 percent of the year's total mine production of ores and concentrates (U.S. Bureau of Mines, 1978b).

At the ore stage, the lead content of the material mined ranges from a high of between 7 and 8 percent to a low of less than 0.01 percent. Lead ores are those in which lead is the predominant nonferrous metal value, as illustrated by the Missouri deposits and selected mines in Colorado and Idaho. Zinc-lead ores contain more zinc than lead, among which the eastern replacement deposits and many western fissure-vein deposits are examples. Complex ores include copper-lead-zinc, silver-lead, copper-lead, and other combinations in which lead is a minor constitutent that usually follows the major nonferrous constituent through several stages of concentration until an economic separation can be made.

The old lead belt in Missouri was averaging a little over 5 percent lead in the hoisted ore in the first part of the 1966-1975 decade. The new lead belt started out at 6.5 to 7.0 percent of lead but was averaging less than 6.7 percent in 1974. Production of ores mined predominantly for lead (i.e., "lead ores") increased from 5.2 million metric tons (5.7 million short tons) in 1966 to a high of 8.4 million metric tons (9.3 million short tons) in 1974, while recoverable lead rose from 148,000 metric tons (164,000 short tons) to 527,000 metric tons (581,000 short tons) in 1974.

In underground mining, access to the orebody is achieved by digging a shaft and underground passage ways in barren rock close to the ore. The ore is then extracted by a mining technique appropriate to the size, location, and outline of commercial ore and the strength and cohesion of the surrounding rock. On the average, lead mines in the United States excavate 1 ton of waste rock for every 17.25 tons of ore sent to the beneficiation plant. The waste rock, over 544,000 metric tons, (600,000 short tons) per year is piled on waste dumps and will contain traces of ore from fringe areas of the orebody removed to gain access to commercial ore. With effective grade control in mining, the waste dump will receive less than 1.0 percent of the ore removed and the average lead content of the dump will be less than 0.1 percent. The waste dump is exposed to rainfall with subsequent leaching and runoff. However, waste rock tends to be coarse, presenting only moderate exposure of surface area to the moisture.

4.2.1.2 Beneficiation--

Coarsely crushed ore is hoisted to ground level and transported by truck or conveyor belt to the mill. Secondary and tertiary crushing are followed by wet grinding to liberate individual mineral species for flotation. Lead, zinc, and copper concentrates can be segregated, with pyrite and the gangue (worthless rock) materials reporting in the tailings. Ores are beneficiated by gravity or froth flotation processes to yield concentrates containing a concentration of 70 to 80 percent of lead, and minor amounts of zinc, copper, and other nonferrous metals. Depending on the type of ore treated and processing conditions, recovery of lead in the form of concentrates may range from over 96 percent to less than 50 percent of mill feed. For lead-dominant ores, some 95 percent of the lead content of the feed reports in the lead concentrate, containing a concentration of lead of 75 percent or better; a further 1 percent is contained in the zinc concentrate, and the balance of 4 percent remains in the tailings. (Hallowell et al, 1973).

Industrywide data are not published on the eventual disposition of the recoverable lead in each class of ore. However, direct losses to tailings from the beneficiation step are believed to be less than 7 percent of the recoverable lead mined annually in the United States, based on a 98 percent recovery from lead ores, 85 percent from lead-zinc ores, and 50 percent from all other ores. (Hallowell, et al, 1973).

Other than accidental spillage or equipment breakdown, physical losses occur as dusts from dry crushing and screening operations or as trace residues left in storage tanks, processing vessels, transfer lines, and equipment. The dusts are evacuated by exhaust systems and separated from the air stream by cyclones, electrostatic precipitators, or wet scrubbers, and added to the flotation conditioning tanks with the finely ground feed. The trace residues usually are flushed to floor drains that discharge to the tailings thickener.

The solids, largely pyrite and inert gangue materials with about 0.2 per cent each of major metals in the ore, are disposed of to large tailings ponds which allow settling of solids and recycle of processing water. From 9.07 million metric tons of ore milled annually, about 8.20 million metric tons of tailings solids are impounded.

Two products -- lead concentrates and zinc concentrates -- are forwarded to appropriate smelters. Zinc concentrates exit from the lead flowchart at this point. Lead concentrates, containing up to 6 percent moisture, are transported to a smelter by conveyor belt for contiguous concentrator-smelter locations, or by truck or railcar for noncontiguous locations. The latter shipments usually are bulk transport in dump trucks or open gondola cars.

4.2.1.3 Smelting and Refining--

This major segment consists of three important processing steps -- sintering, smelting, and refining. The sintering step converts lead sulfide to lead oxide with the potential of recovering sulfur dioxide as by-product sulfuric acid. The smelting step reduces lead oxide to elemental lead (lead bullion) containing most of the metallic impurities that were in the concentrate. The refining step separates refined lead (99.99 percent purity) from the metallic impurities, which report in various drosses.

After analysis, lead concentrates are blended with fluxes and recycled slags, dusts, and sinter fines. The blend is loaded onto a continuous sinter belt, ignited, and burned by oxygen in the air. During the initial sintering stages, the strong offgases contain 6 to 8 percent SO_2 which is diverted to the sulfuric acid plant. Weak offgases from sinter cooling stages are piped to the smelter baghouse for recovery of any dust. Cooled sinter is crushed and separated into a coarse stream and a recycle fines stream. Between 30 and 50 percent of the sinter feed consists of recycled material to promote suitable porosity on the belt, and to dilute the sulfide content sufficiently to prevent the attainment of excessively high temperatures which would cause fusion during sintering.

The gases from the sintering operation, averaging about 4 percent SO_2 are passed through a gas-cleaning system to remove particulates and then to a contact sulfuric acid plant. While the gas-cleaning effectively removes the metal-containing dusts there is sufficient carry-over of volatile organics from flotation reagents to produce a dark color sulfuric acid, usually sold at a discount, principally for use in making fertilizer.

The coarse sinter is conveyed to the smelting operation where it is combined with coke and recycled refinery drosses and charged intermittently to a blast furnace where the coke ignites, and heats the sinter to incipient fusion, and provides the reducing gas that converts lead oxide to metallic lead. Fluxes in the sinter become slag which is tapped from the bottom of the furnace with molten lead to a settler. Lead overflows a weir in the settler and is transferred in ladles to the refinery or cast into cakes as lead bullion. Slag overflows to an inclined iron trough where it is granulated by water jets for recycle, as a diluent, to the sinter plant or for discharge to a dump. Offgases from the furnace pass through a baghouse for dust removal before discharge to the atmosphere. Baghouse dust is recycled to the sinter preparation area.

Although all the lead smelters in the United States are essentially based on the same process, the western smelters employ more complicated flow sheets because of the more complex nature of the western concentrates, complex imported materials, and their interrelationship with copper and zinc plants. For example, the slag in Missouri is sufficiently low in zinc to discard, but at Western smelters, slag is treated for zinc recovery in a slag fuming furnace. (U.S. Environmental Protection Agency, 1975a).

Lead from the southeast Missouri lead belt is relatively free of arsenic, antimony, and bismuth and is usually refined immediately after smelting. Although there are variations, the refining process is essentially as follows. Molten lead from the smelter is decoppered by stirring with soda ash, sand, coke, and recycled sulfur dross while cooling to 510 C (950 F). The resulting dry dross is skimmed off and smelted in a reverberatory furnace yielding copper matte, lead bullion, and a small amount of slag. The cooled lead is stirred with sulfur to complete decoppering, the dross being recycled to the prior step. Next, the molten lead is desilvered in two stages using recycled zinc-silver skim and fresh zinc powder. After a number of recyclings, the zinc-silver skim contains over 15 percent silver and is treated to distill off the zinc while the impure silver is cast into bars for shipment to a silver refinery. After desilvering, the still molten lead is subjected to a vacuum in which the residual zinc distills. The resulting 99.99 percent pure lead is cast into pigs for shipment as refined lead (U.S. Environmental Protection Agency, 1975b).

Lead from western lead-zinc and complex ores contains significant amounts of arsenic, antimony, and bismuth in addition to copper, zinc, silver, and gold. Lead bullion from a smelter -- melted if necessary -- is stirred with soda ash and coke while cooling to precipitate copper in a lead dross treated, as before, in a reverberatory furnace yielding copper matte, lead bullion, and slag. Decoppered lead is given two sequential Harris process treatments (molten caustic soda) to remove firstly, arsenic and tin, if any, and secondly, antimony. The resulting lead bullion is further decoppered by stirring with sulfur, then desilvered and dezinced as described above. Then, the dezinced lead is stirred with a mixture of calcium and magnesium metals which form intermetallic compounds with the bismuth impurity. Finally, dissolved calcium and magnesium are removed by treating the lead with caustic soda. The refined lead is cast into pigs for sale or the preparation of lead alloys. Harris process slags are treated to recover arsenic and antimony as the oxides. Calcium-magnesium-bismuth slags are treated with chlorine to recover commercially pure bismuth. (U.S. Environmental Protection Agency, 1975b).

4.2.1.4 Production and Products--

Primary lead smelting and refining increased in the 1966-1975 decade, in keeping with the growth of domestic mining. In 1966, four smelters, two smelter-refiners, and four refineries produced 428,000 tons (472,000 short tons) of refined lead. In the following 10 years, two smelters and one refinery were deactivated while two refiners switched solely to secondary production, but two new smelter-refineries were built and a third expanded extensively. Thus, refined lead output in 1972 (the peak year) was 631,000 metric tons (696,000 short tons), an increase of 47 percent, equivalent to an annual compound rate of nearly 4 percent.

Locations of the present primary lead smelters and refineries are shown in Figure 4.2. Pertinent information on their capacities and characteristics are contained in Table 4.2.

U.S. production of refined lead and antimonial lead during the 1966-1975 decade is shown in Table 4.3. There was a dramatic increase in primary lead production from domestic areas, beginning in 1969:

Year	Thousands of Metric Tons	Year	Thousands of Metric Tons
1965	273	1972	562
1966	297	1973	547
1967	288	1974	602
1968	326	1975	564
1969	462	1976	528(e)
1970	519	1977	488(e)
1971	525		

This coincided with the commissioning of the first mine in the "new lead belt", following the discovery of massive replacement deposits in southeastern Missouri in geological formations different from those containing the deposits opened up in 1763. Since that time the "new lead belt" has dominated United States production. Production has ceased at the mines in Kansas and Oklahoma, and declined significantly from the fissure-vein deposits in the western states. Production from the eastern replacement deposits continues to contribute only minor tonnages.

Lead bullion is the designation given lead as produced from the blast furnace. While lead from Missouri ores is generally pure enough for most commercial uses without sophisticated refining, and is termed "chemical lead", that from western and most foreign ores contains enough gold and silver to make their extraction profitable.

Other metals, especially antimony, must be reduced to very low levels to have "soft" lead, which is easily workable and the type used for most applications. Antimonial lead, containing as much as 8 percent antimony, is

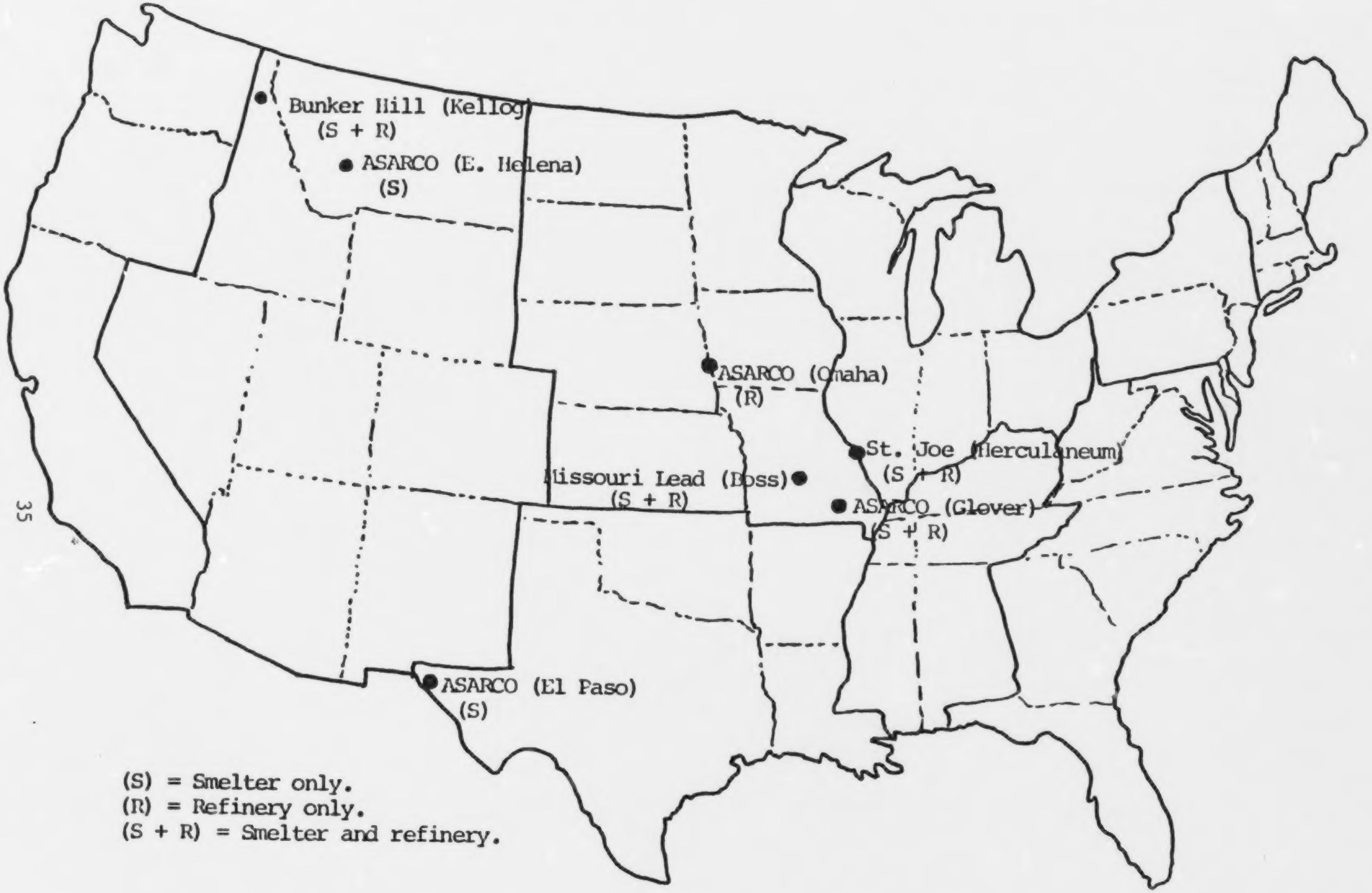


Figure 4.2. Geographic locations of domestic primary lead smelters and refineries
Source: U.S. Environmental Protection Agency (1975a)

TABLE 4.2. LEAD SMELTERS AND REFINERIES[a]

Company	Location	Annual Tons of Lead Containing Material Treated 1972	First Year Of Operation	Raw Materials Used	Slag Treatment	Acid Plant	Products
American Smelting and Refining Company	Glover, Missouri	159,600	1968	Lead Conc. Waelz Residue Lead Residues	None	None	Refined Lead Copper Dross Retort Bullion
	East Helena, Montana	192,000	1888	Lead Conc. Siliceous Ores Zinc Residue	Slag Fuming Furnace	None	Lead Bullion Soda Ash Matte Soda Ash Speiss Lead Baghouse Dust Zinc Fume
	El Paso, Texas	not reported	1887		Slag Fuming	None	Lead Bullion Zinc Fume
	Omaha, Nebraska	136,000	1870	Lead Bullion Secondary Lead	None	None	Refined Lead Antimonal Lead Solder Misc. Lead Alloys Bismuth Copper Matte Sodium Telluride Slag
Bunker Hill Co.	Kellogg, Idaho	550,000	1918	Lead Conc.	Slag Fuming	300 TPD	Refined Lead, Gold, Silver, Antimony
St. Joe Minerals	Herculaneum, Missouri	336,000	1892	Lead Conc.	None	300 TPD	Refined Lead, Silver Bullion, Copper Matte
Missouri Lead Operating Co.	Boss, Missouri	192,500	1968	Lead Conc.	None	225 TPD	Refined Lead Copper Matte Dross Silver Bullion

[a] Source: U.S. Environmental Protection Agency(1975a).

TABLE 4.3. U.S. PRODUCTION OF REFINED LEAD AND ANTIMONIAL LEAD AT PRIMARY REFINERIES, BY SOURCE MATERIAL, 1966-1975[a]

	Production, Thousands of Metric Tons									
	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975
Refined Lead										
From primary sources:										
Domestic ores and base bullion	289	235	317	466	479	520	524	515	526	481
Foreign ores and base bullion	111	110	107	113	126	70	94	97	84	96
From secondary sources	8	2	2	5	4	1	1	--	--	--
	408[b]	347	426	584	609	591	619	612	611	577
Antimonial Lead										
From primary sources:										
Domestic ore	5	5	14	11	8	11	6	8	5	2
Foreign ore	5	3	3	4	3	4	2	4	4	--
From secondary sources	10	7	6	6	7	2	5	1	1	3
	20	15	24	21	17	17	13	13	10	5
Total Primary Sources	410	353	442	594	616	604	625	624	620	579
Total Secondary Sources	18	9	8	10	11	3	6	1	1	3
GRAND TOTAL	428	362	450	605	627	607	631	625	621	582

---

[a] Source: U.S. Bureau of Mines Minerals Yearbooks.

[b] Totals may not equal sum of figures shown because of rounding.

a specialty lead favored for battery use. Standard specifications for pig lead, as specified by ASTM Designation B29-55, are shown in Table 4.4.

4.2.2 Secondary Lead

Lead is a durable metal and is one that is relatively easy to refine or rerefine. There is, accordingly, a major secondary market, comparable in size to the primary metal market. Secondary lead is recovered from scrap, wastes, drosses, and residues. Because of its excellent resistance to corrosion, many lead products remain virtually unchanged during their lifetime, and recovery is not difficult. The chief source of secondary lead is from automobile storage batteries that have been scrapped after 3- to 4-year service. In the U.S. about 80 percent of the lead used in the manufacture of storage batteries is recycled; in some foreign industrial countries the percentage is higher. An indication of the flow of scrap is given by Figure 4.3. (Ryan and Hague, 1976).

In the recycle industry scrap is classified into two categories, "new" or prompt industrial scrap, and obsolete or old scrap. "New" scrap is that resulting from metal industry operations, e.g., punchings, borings, trimmings, imperfect products, etc. It is easily kept segregated with respect to composition, and finds a ready market. A recent study (Battelle-Columbus, 1972) estimated that, for most metals, recycle of "new" scrap approaches 100 percent of that generated.

Obsolete scrap, or old scrap, consists of metal products (industrial or consumer) that have been in use, served their purpose, and been discarded. For lead, obsolete scrap in recent years accounts for about 85 percent of total scrap, as illustrated by Table 4.5. The largest source of obsolete scrap is battery-lead plates, and accordingly, the principal recovered product is antimonial lead.

The breakdown of scrap receipts is illustrative of the general origins of lead scraps. Another annual tabulation of the U.S. Bureau of Mines is of stocks and consumption of new and old scrap; the 1975 figures are illustrative of the origins and relative significance of the various types of scrap:

	Scrap Receipts, Metric Tons, Gross Weight	
	New Scrap	Old Scrap
Soft Lead	--	30,430
Hard Lead	--	24,610
Cable Lead	--	44,140
Battery-Lead Plates	--	561,180
Mixed Common Babbitt	--	2,970
Solder and Tinny Lead	--	9,920
Type Metals	--	17,450
Drosses and Residuals	121,320	--
Total	121,320	690,700

TABLE 4.4. STANDARD SPECIFICATIONS FOR PIG LEAD (B29-55)[a]

	Corroding Lead[b]		Chemical Lead[c]		Acid Copper Lead[d]		Common Desilverized Lead[e]	
	Max	Min	Max	Min	Max	Min	Max	Min
Silver	0.0015	--	0.020	0.002	0.002	--	0.002	--
Copper	0.0015	--	0.080	0.040	0.080	0.040	0.0025	--
Silver and copper	0.0025	--	--	--	--	--	--	--
Arsenic, antimony and tin, together	0.002	--	0.002	--	0.002	--	0.005	--
Zinc	0.001	--	0.001	--	0.001	--	0.002	--
Iron	0.002	--	0.002	--	0.002	--	0.002	--
Bismuth	0.050	--	0.005	--	0.025	--	0.150	--
Lead (by difference)		99.94		99.90		99.90		99.85

[a] Source: Annual Book of ASTM Standards, 1978. Reprinted by permission of the American Society for Testing and Materials, (C) 1971.

[b] Corroding lead is a designation that has been used in the trade for many years to describe lead which has been refined to a high degree of purity.

[c] Chemical lead has been used in the trade to describe the undesilvered lead produced from Southeastern Missouri ores.

[d] Acid-copper lead is made by adding copper to fully refined lead.

[e] Common desilvered lead is a designation used to describe fully refined desilvered lead.

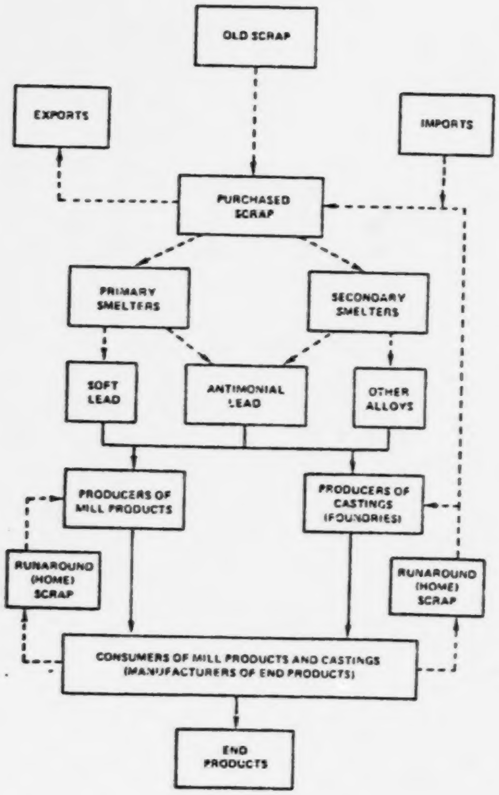


Figure 4.3. Lead scrap flow.

Source: Ryan and Hague (1976).

TABLE 4.5. LEAD RECOVERED FROM SCRAP PROCESSED IN THE UNITED STATES, 1966-1975[a]

	Thousands of Metric Tons									
	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975
Kind of Scrap										
New Scrap[b]										
Lead-base	72	65	67	74	79	93	103	100	84	82
Copper-and tin-base	7	5	5	6	4	4	5	4	4	3
Total	79	70	72	80	83	97	108	105	88	85
Old Scrap										
Battery lead plates	259	275	281	317	318	302	315	335	379	379
All other lead-base	163	141	130	134	126	129	122	140	152	123
Cooper-and-tin base	18	16	16	17	15	13	14	13	13	9
Total	440	432	427	468	459	444	451	488	545	511
Grand Total	519	502	499	548	542	541	559	592	633	596
Form of Recovery										
As soft lead	142	136	126	140	144	136	157	169	206	246
In antimonial lead	257	262	280	311	316	311	314	341	337	286
In other alloys	121	104	94	97	82	94	88	84	81	65
Total	520	502	500	548	542	541	541	594	634	597

[a] Source: U.S. Bureau of Mines Annual Minerals Yearbooks.

[b] New Scrap consists primarily of drosses and residues.

Scrap receipts are not directly comparable to lead recovered, being on a gross weight basis inflated by an unknown amount, since substantial amounts of non-lead materials are included in the gross weights.

According to the U.S. Bureau of Mines (1976) in 1975 approximately 115 secondary lead plants were engaged in recovering lead and lead alloys from recycled scrap material; five plants closed during the year. Important secondary smelters are located in major metropolitan areas, such as New York, Philadelphia, Baltimore, Cleveland, Chicago, Baton Rouge, Dallas, Los Angeles, and San Francisco. Lead-bearing scraps of copper or tin alloys, and selected lead alloys usually are merely melted and adjusted for proper alloy composition.

4.2.3.1 Scrap Pretreatment--

Because obsolete scrap makes up 85 percent of the lead scrap consumed, it can be obtained from a wide variety of sources. These scraps may contain organic materials such as insulation, grease, and oil. The major metallic elements found in the scrap are antimony, tin, arsenic, copper, cadmium, zinc, silver, indium, tellurium, and bismuth.

Lead scrap must be treated by densification and/or partial removal of metallic and nonmetallic contaminants in order to make it more amenable for further processing. Large pieces of lead-bearing scrap such as drosses, residues, and slags must be reduced in size before further processing. These are crushed in a jaw crusher to the desired size for processing to recover the lead content. Handling and crushing of these scrap materials gives rise to dust emissions which need to be controlled.

A number of relatively impure lead scraps (tooling dies, type metal drosses, lead sheathed cable and wire, and lead drosses and skimmings from a number of sources) are usually given a pretreatment to recover the lead values. One process is the rotary/tube sweating route where the scrap is charged to a furnace, the lead melted from the remaining materials, and the molten lead poured from the furnace, leaving the higher-melting residues behind. The sweated lead normally is given another furnace treatment to refine or alloy the recovered lead into an acceptable product.

Reverberatory sweating is used to treat lead scrap with a higher lead content than that charged to a rotary/tube furnace. The charge materials usually are battery plates, lead oxides, drosses, lead scrap, and certain residues. The process steps are the same as for rotary tube sweating. That is, charging the solid material to the reverberatory furnace, melting, and pouring the lead away from the solid residue.

Zinc-bearing scraps will produce a zinc-containing dust, and the dust collected by the baghouse may be leached with sulfuric acid to remove and recover the zinc. The lead-bearing residue is returned to the blast furnace for refining.

4.2.2.2 Smelting--

Smelting is commonly used to purify scrap by removing some of the metallic and nonmetallic contaminants. The scrap may consist of new scrap, pretreated scrap, type metals, babbit metals, mixed scrap, metallics from batteries, or flue dusts. The secondary lead industry is basically the recovery of lead from automobile batteries; in 1975 almost 69 percent of the lead scrap consumed was in the form of obsolete, discarded automobile batteries. In this process any liquid remaining in the battery is drained out, the battery is crushed to break the lead and lead compounds from the nonlead portion, and the mixture screened to separate the lead from the organic materials that form the battery case. The recovered lead scrap can then be fed into a reverberatory furnace or blast furnace for further processing. The smelting scheme is illustrated by Figure 4.4 (U.S. Environmental Protection Agency, 1977a).

Soft lead is recovered by reverberatory smelting. Because of the low melting point of lead, it rapidly separates from the other metals. The molten material is agitated by bubbling air or steam through it to oxidize impurities such as antimony that then rise to the surface and enter the slag. The slag, containing a mixture of lead oxides and antimony, is charged to the blast furnace for further processing. The fairly pure molten lead is poured into ingots.

Particulate matter consisting of metallic oxides, sulfides, and sulfates of lead, tin, arsenic, copper, and antimony is collected by passing the flue gases through a baghouse and recycling the dust to the furnace for lead recovery.

In blast furnace smelting a hard antimonial lead is produced by charging a mixture of pretreated scrap, reverberatory slag, battery scrap, drosses and rerun slag to the furnace along with coke and scrap iron and heating. The lead and antimony from the slags are reduced to the metallic state by carbon monoxide produced by combustion of the coke. The molten lead is withdrawn almost continually from the furnace; the slag is tapped at intervals. As the material in the furnace melts down and is tapped, additional charges, consisting of 4.5 percent scrap iron, 3 percent limestone, 5.5 percent coke, 4.5 percent rerun slag, and 82.5 percent drosses, oxides, reverberatory slag, and residues are added. The molten lead is cast into ingots or transferred to refining kettles.

4.2.2.3 Refining--

Intermediate products such as soft lead and antimonial lead from the blast furnace and reverberatory furnaces require further refining to obtain final lead products. Some lead oxide materials are made in addition to metal products.

In kettle (softening) refining, hard lead, either cast or molten, and fluxes are charged to a preheated kettle and melted. After mixing the flux into the molten charge, the drosses are skimmed off and the metal cast into

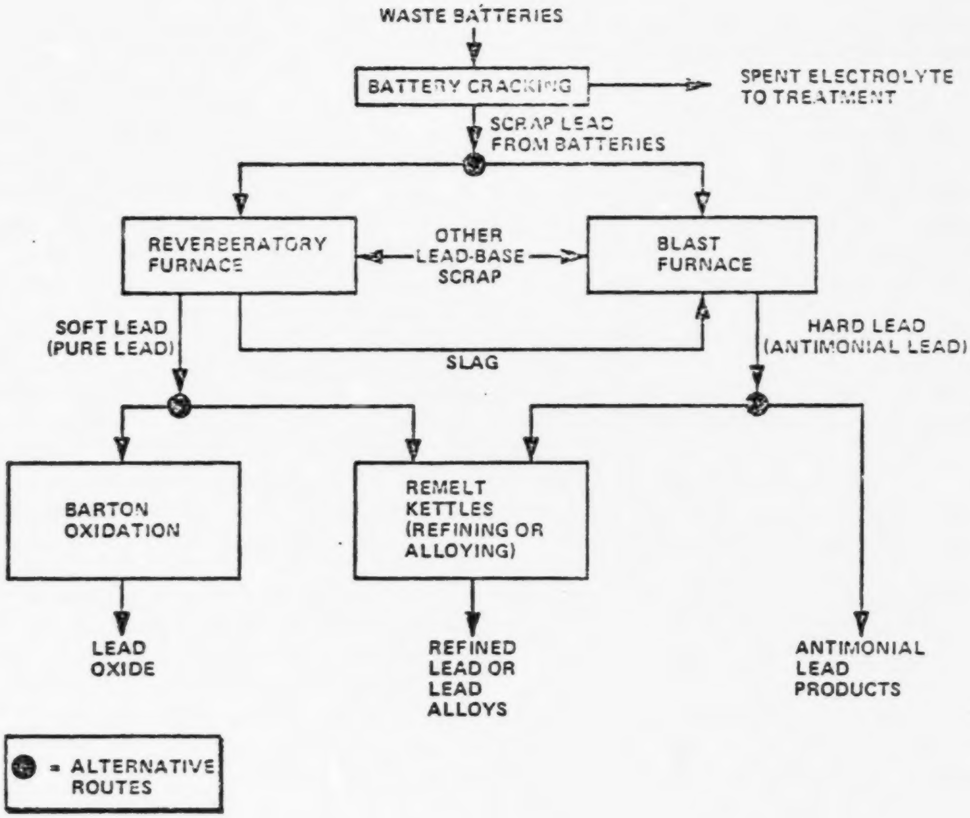


Figure 4.4. Secondary lead smelting process
Source: U.S. Environmental Protection Agency (1977a).

ingots. Several fluxes, including sodium hydroxide, sodium nitrate, aluminum chloride, aluminum, sawdust, and sulfur, are added to remove deleterious metals, including antimony. These metals are removed in the dross. The solid wastes contain skimmings, flux, and metals separated from the melt during refining. These wastes are also returned to the blast furnace for lead recovery.

Kettle (alloy) refining is the process of adjusting the metal composition of reverberatory or blast furnace lead to produce the desired lead alloy. The process is essentially the same as in the previous process. The charge is melted, flux stirred in, the alloying agents added, the skimmings removed, and the molten lead alloy cast. Antimony, copper, tin, and silver are common alloying agents.

The kettle oxidation process is used to produce battery lead oxide, which is essentially lead monoxide (PbO) containing about 20 percent of lead metal. The operation consists of charging molten led to a kettle, agitating the molten material rapidly while air is passed over the melt, drawing the air through a duct to a baghouse, and collecting the lead oxide plus lead metal product

Reverberatory oxidation is used to produce either lead monoxide or red lead (Pb_3O_4). PbO is made by oxidizing lead under careful control. Red lead results from overoxidizing the lead. The process involves charging the hot furnace with molten lead, agitating and simultaneously oxidizing the molten lead with air, and removing the lead oxide from the furnace.

4.2.3 Supply/Demand Relationships

Estimating the total supply of lead available for United States consumption each year is an almost impossible task to accomplish with accuracy. The analysis is complicated by the varying percentages of lead in the multiplicity of lead-containing materials and lead scraps in varying stages of processing by domestic producers, and further compounded by the imports and exports of these same materials. However, by focusing on the main factors and neglecting the minor aberrations such as the above a close approximation can be derived. This can be done by defining the total supply as the total of primary and secondary production plus imports, adjusted for the net change in producers and consumers stocks. On this basis, the total supply of lead in the United States varied during the years 1967-1977 as shown in Table 4.6, from a minimum of 1,163,000 metric tons in 1967 to a maximum of 1,497,000 metric tons in 1977. It will be noted that there has been a gradual, if irregular, decline in imports as the new rich Missouri deposits were exploited and began to affect market balance. There was, however, an increase in imports in 1977. Total imports of all types of lead-containing material during this same period are shown in Table 4.7. Concurrently, exports of refined lead and lead scrap materials were inconsequential until 1972 when world demand surpassed supply. The continuing world shortage during 1973 and early 1974 provided unusual opportunities for the American producers to penetrate foreign markets at a profit. By 1975, the worldwide economic stagnation had eliminated much of

TABLE 4.6. SUPPLY OF LEAD IN THE UNITED STATES, 1967-1977[a]

		Thousands of Metric Tons			
Year	Primary Production	Secondary Production	Imports[b]	Net Stock Change[c]	Total Supply
1967	353	503	330	(23)	1163
1968	442	500	307	47	1296
1969	594	548	253	(44)	1351
1970	616	542	222	(89)	1291
1971	604	542	177	72	1395
1972	625	560	220	(15)	1390
1973	624	594	162	45	1425
1974	620	634	107	(67)	1294
1975	579	596	91	(2)	1264
1976	598	645	135	107	1485
1977	555	648	244	44	1497

[a] Source: U.S. Bureau of Mines Annual Minerals Yearbooks, (1966-1976) U.S. Bureau of Mines Mineral Industry Surveys, August, 1977; March, 1978.

[b] Imports for consumption of pigs and bars (lead content).

[c] Includes stocks at both producers' and consumers' plants. Figures in parentheses, (), indicate net addition to inventory.

TABLE 4.7. U.S. IMPORTS OF LEAD BY TYPES OF MATERIAL, 1967-1977[a,b]

	Thousands of Metric Tons of Lead Content				
Year	Ore, flue dust and matte	Base Bullion	Pigs and Bars	Reclaimed Scrap, etc	Total[c]
1967	113	1	330	9	453
1968	80	<1	307	6	393
1969	99	2	253	6	360
1970	102	<1	222	6	330
1971	60	<1	177	3	240
1972	92	1	220	3	317
1973	100	<1	162	3	264
1974	86	1	107	1	195
1975	79	<1	91	5	176
1976	69	2	132	4	207
1977	66	7	237	6	316

[a] Source: U.S. Bureau of Mines Minerals Yearbooks (1967-1976)
U.S. Bureau of Mines Mineral Industry Surveys, August, 1977; March, 1978.

[b] Does not include imports of pigments and lead compounds.

[c] Totals may not equal sums of figures shown because of rounding.

the foreign premium price for lead and U.S. exports were returning to traditional levels (Table 4.8). Historically, much of the scrap exports consisted of contaminated dross and baghouse dusts requiring extensive processing to recover the valuable metals.

Long-term trends in the lead industry are illustrated by Figure 4.5, in which the total of annual primary and secondary production is compared to total annual consumption. Approximate balance of supply and demand was achieved by imports, which normally ranged between about 175,000 and 350,000 metric tons per year, except for a few high years in the early 1950's. Secondary production is less erratic and more stable than primary production, but has also exhibited a steady upward trend for the last two decades. During most of the 1960's, secondary lead production exceeded primary production; in fact, it was not until the "new lead belt" mines in Southeastern Missouri came into production that primary production regained the lead.

Employment in the lead producing industry has held fairly constant over the last five years although decreasing slightly. Mine and mill employment has gradually decreased from 4,900 in 1973 to 4,700 in 1977; smelter and refinery employment decreased only from 2,450 to 2,400 during the same period (U.S. Bureau of Mines, 1978b).

4.2.3.1 Stockpiling--

During World War II and again in 1951-1952, the government regulated the lead industry to provide adequate supplies for all essential purposes. Stocks were reduced during World War II, but stockpiling was resumed in 1950, and by the end of 1958 the domestic purchase program was completed. Disposal of excesses began in 1964. The government stockpile had a substantial impact on the lead industry, particularly during 1972-1974, when 473,500 metric tons of stockpile excess was sold. In 1974, withdrawals from the stockpile provided approximately 14 percent of the new supply of lead. (Ryan and Hague, 1976). Actual physical drawdown of government stocks was only about 6,300 metric tons during 1975, a year of depressed demand for lead. There were no withdrawals in 1976 and 1977 and stockpile status, as of November 30, 1977, was 545,700 metric tons, with a goal of 785,000 metric tons, 865,000 short tons, and none authorized for disposal (U.S. Bureau of Mines, 1978b).

4.2.3.2 Refined Lead Prices--

In the early part of the 1966-1975 period, common pig lead was priced for marketing at either St. Louis (Missouri) or New York (New York). Starting in 1972, the quotation basis is "delivered common pig lead" with New York as the site for posting. The London price is that derived from daily trading on the London Metal Exchange, the accepted indicator of world pricing trends. London lead prices traditionally are lower than those in the United States. Frequently, the difference is enough to cover ocean freight and insurance costs plus a little extra for the import agent, a matter of 2 to 2.5 cents per pound. This relationship was maintained from

TABLE 4.8. U.S. EXPORTS OF LEAD BY TYPE OF PRODUCT, 1967-1977[a,b]

Year	Thousands of Metric Tons				
	Pigs, Bars, and Anodes				
	Unwrought Lead and Alloys	Wrought Lead and Alloys	Total	Scrap	Grand Total[d]
1967	--	--	5.9[c]	0.4	6.3
1968	--	--	7.5[c]	0.9	8.4
1969	2.3	2.2	4.5	2.1	6.6
1970	4.5	2.5	7.0	3.8	10.9
1971	3.4	2.0	5.4	15.5	20.9
1972	6.0	1.6	7.6	32.0	39.6
1973	47.1	13.4	60.4	54.3	114.7
1974	48.6	7.6	56.2	53.9	110.1
1975	16.7	2.6	19.3	45.3	42.5
1976	--	--	5.3	42.5	47.8
1977	--	--	8.9	77.5	86.4

[a] Source: U.S. Bureau of Mines Minerals Yearbooks (1967-1976)
U.S. Review of Mines Mineral Industry Surveys, August, 1977; March, 1978

[b] Does not include exports of lead compounds.

[c] Prior to 1969, pigs, bars, and anodes not subdivided.

[d] Totals may not equal sums of figures shown because of rounding.

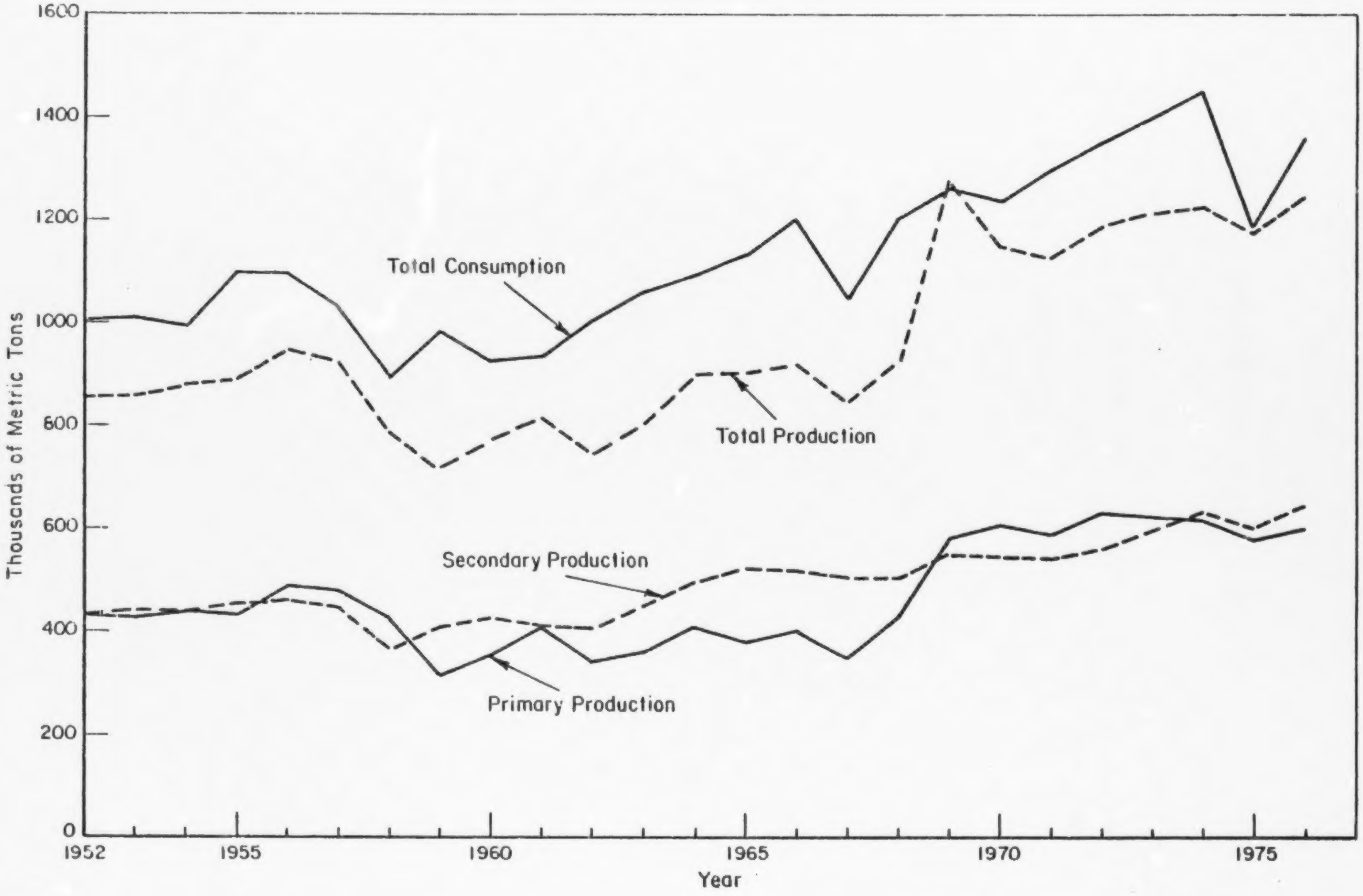


Figure 4.5. Trends in the lead industry in the United States.

Source: U. S. Bureau of Mines Minerals Yearbooks.

1966 until the early part of 1973 when worldwide shortages drove London prices above those in the United States.

Refined lead has the status of a commodity on world markets and its price is quite responsive to shortages or surpluses, both short term and long term. Lead prices trended downward in the 1966-1968 period under the increasing pressure of surplus production, largely overseas. Prices firmed in 1969 and early 1970 until the slowdown in the U.S. economy triggered a period of excess stocks well into 1971. After reasonable balance in 1972, booming economies worldwide in 1973 and 1974 liquidated producers' and dealers' stocks and the London price exceeded the United States price, creating a good export market for the new production in southeastern Missouri. The worldwide downturn in 1975 was reflected in falling prices and a return to the traditional spread between U.S. and London quotations. However, production costs for the "new Missouri lead belt" tend to maintain a floor price under U.S. quotations at 19 cents per pound, 5 cents higher than the lowest quote in 1972, the last "normal" year.

Since 1975, additional increases in the U.S. quotations have been introduced without regard to the status of producers' or dealers' stocks. The average U.S. producer price in January-March, 1978, for common lead was 33.0 cents per pound, a 2 cent increase above mid-1977 prices, and a full 10 cents above the average 1976 price. (U.S. Bureau of Mines, 1978a)

4.2.3.3 Reserves--

New discoveries since 1965 have substantially increased the United States resource base for lead, and annual mine production has more than doubled.

According to the latest U.S. Bureau of Mines estimates, United States reserves are an important part of world totals:

Region	Lead Content, Thousands of Metric Tons
United States	25,800
Australia	17,000
Canada	11,700
Mexico	4,100
Peru	3,200
Other Latin American Countries	4,100
Other Market Economy Countries	27,600
Central Economy countries	30,000
	123,500

U.S. lead reserves represent a decrease from previous estimates because certain inferred reserves not having near-term potential have been excluded. (U.S. Bureau of Mines, 1978b).

Including hypothetical undiscovered economic and some identified marginally economic resources doubles the world total. If speculative and economically submarginal lead deposits as well as undiscovered lead deposits in new areas of the world are considered, then the total resource of lead may be as high as 1.5 billion tons (Ryan and Hague, 1976).

Ryan and Hague estimated the distribution of United States reserves by states as follows:

States	Lead Content, Millions of Metric tons
Missouri	46.9
Washington, Idaho, Montana	4.5
Colorado, Utah, New Mexico, Arizona, California	1.6
Virginia, New York, Maine, Wisconsin, and others	0.5
	53.5

In the opinion of the U.S. Bureau of Mines (Ryan and Hague, 1976) the United States resource base is more than adequate to support the domestic component of primary lead demand to 2000.

4.3 LEAD CONSUMING INDUSTRIES

The consumption of lead in the United States is inextricably related to the automobile industry. During the last several years from 800,000 to 1,000,000 metric tons of lead, accounting for from 60 to over 70 percent of total annual lead consumption, have gone into storage batteries and gasoline antiknock additives. Lead-acid storage batteries, as described later, represent the only large segment of the lead industry with significant growth potential. Traditional lead-based metal products and pigments face declining or stagnant rather than growing markets, and the use of lead in gasoline is mandated to be drastically reduced within the next several years. The world consumption of lead is slowing, and Bank of America economists predict consumption to grow at only a 2 per cent rate through 1985, compared with a 3 per cent annual growth rate during the past 20 years (Anonymous, 1976).

Lead and lead compounds have many other smaller, but nevertheless important uses. Metallic lead is used for ammunition, bearing metals, cable covering, pipe caulking, collapsible tubes, foil, solder, type metal, weights and ballast, radiation attenuation, and in sheet form, for attenuation of noise or vibration.

Lead compounds also have a wide diversity of uses. There are scores of inorganic compounds and over 1000 organolead compounds. Some illustrative uses of lead compounds are listed in Table 4.9, although some of the possible use shown are minor or insignificant.

U.S. lead consumption, grouped into five broad classes of products, is shown in Table 4.10 for the 1967-1976 decade. As is evident in this tabulation, storage batteries, an essentially nondissipative use, account for over half of annual lead consumption. Most of the remaining lead uses would have to be classified as dissipative, either totally and prompt, such as gasoline antiknock additives, or totally and slowly, such as pigments in paint. A smaller third class would include the mostly metallic uses, where the lead is in a reclaimable form, but which may or may not be recovered.

A more detailed breakdown of consumption uses for 1973-1976, the most recent years for which data are available, is shown in Table 4.11. The various use categories shown are discussed in the following sections.

These data are derived from U.S. Bureau of Mines sources. The Bureau of Mines has for a number of years compiled probably the most complete lists of lead consumption in the United States by product. The classifications used have been consistent in that they provide continuity, but the breakdown in some cases is rather broad, so that some important end uses of compounds do not show as line items. An example of this is the class of "red lead and litharge" under pigments. Litharge (PbO) has many uses other than as a pigment. A signficant fraction of the litharge reported is used in the manufacture of batteries; a new significant use is the preparation of a number of lead compounds used as stabilizers in plastics and elastomers. Nevertheless, the Bureau of Mines breakdown of lead use by products provides the best overall picture currently available of the relative importance of most lead uses.

4.3.1 Storage Batteries

Storage batteries represent by far the largest single category of lead use. Approximately 93 percent of the storage batteries produced in this country are classified as SLI (starting, lighting, and ignition) and are used for equipping new cars or as replacement batteries for existing vehicles. The remaining 7 percent are industrial batteries used as emergency power sources, to power industrial and mining vehicles, in submarines, etc. Although battery design and manufacture have been improved to extend the useful life of batteries, expansion in the number of automobiles on the road and the development of new transportation modes--i.e., industrial lift trucks, golf carts, snowmobiles, etc.--has broadened the markets for batteries (Short and Associates, 1976).

4.3.1.1 Manufacturing--

The manufacturing process for making storage batteries is complex, but a simplified version follows. The battery grids are cast, then filled with a lead oxide (black oxide) paste to form a porous lead oxide plate, and cured to increase strength. The plates and separators are assembled into elements and placed in the battery case. The elements are welded together,

TABLE 4.9 SELECTED LEAD COMPOUNDS AND THEIR USES[a]

Chemical Compounds	Uses
Lead acetate	Dyeing and printing cottons Manufacture of lead salts Astringent (veterinary) Paint drier Lead coating of metals
Lead alloys	Slush coatings Type metals for printing Batteries Battery grids Bearing metals Solders Fusible alloys Communication cables Pipe and sheet in chemical installations Corrosion protection coatings
Lead antimonate	Pigment Staining glass Crockery and porcelain
Lead arsenate	Insecticides Veterinary medicine
Lead azide	Primer in explosives
Lead borate	Paint drier and production of conductive coatings Glass Pottery Porcelain Chinaware
Lead carbonate	Paints Ceramics Glazes Processing of parchment
Lead chloride	Manufacturing of white lead dyes Solder and flux
Lead chromate	Pigment Printing of fabrics Decorating china and porcelain

TABLE 4.9 SELECTED LEAD COMPOUNDS AND THEIR USES[a] (Cont.)

Chemical Compounds	Uses
Lead cyanamide	Corrosion inhibitors (Antirust paints)
Lead dioxide	Electrodes Dyes Rubber substitutes Manufacture pigments Analytical purposes
Lead fluosilicate	Electrolyte in electrolytic refining of lead
Lead formate	Rubber compounding
Lead fumarate	Plastisols Phonograph records Electrical insulation
Lead iodide	Bronzing, printing Photography
Lead linoleate	Driers in paints
Lead molybdate	Paint Pigment
Lead monoxide, litharge	Ointments Plasters Glaze Flux Pigments Analytical Driers
Lead naphthenate	Driers in paint
Lead nitrate	Match industry Pyrotechnics Chemical intermediates
Lead oleate	Driers in paint

TABLE 4.9 SELECTED LEAD COMPOUNDS AND THEIR USES[a] (Cont.)

Chemical Compounds	Uses
Lead orthophosphate	Stabilizer for plastics Pigments Anticorrosive agents
Lead perchlorate	Analytical reagent
Lead phosphate	Stabilizer for plastics
Lead selenide	Semiconductor Infrared detector
Lead silicate	Glass, ceramics
Lead stannate	Ceramics
Lead styphnate	Explosives detonator
Lead subactate	Sugar analysis
Lead sulfate	Pigments Batteries Lithography Fabrics
Lead sulfide	Photoelectric cell Infrared detector Photosensitive resistor circuits
Lead telluride	Photoelectric cell Infrared detector Photosensitive resistor circuits
Lead tetroxide, red lead	Pigments Dyes Chemicals Matches Pryotechnics Curing agents (rubber substitute)
Lead tetramethyl	Antiknock Fungicide Filling of Geiger counters

TABLE 4.9 SELECTED LEAD COMPOUNDS AND THEIR USES[a] (Cont.)

Chemical Compounds	Uses
Trialkyl or alkyl aryl lead	Polymerization Catalysts Stabilizers for PVC Electroplating Lubricating oils Biocidal agents
Lead thiocyanate	Primer in explosives Matches Dyes
Lead thiosulfate	Rubber accelerator Lead mirrors
Lead titanate	Pigment Ceramics
Lead tungstate	Pigment
Lead vanadate	Pigment
Lead zirconate	Pigment

[a] Source: Lutz, et al.,(1970)

TABLE 4.10. U.S. LEAD CONSUMPTION BY CLASS OF PRODUCT, 1967-1976[a]

Product	Consumption, Thousands of Metric Tons									
	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976
Metal products	331	331	322	287	333	307	308	298	240	238
Storage batteries	423	466	529	539	617	659	698	773	635	746
Pigments[b]	92	97	92	89	73	81	99	105	72	96
Chemicals[c]	225	238	247	253	240	253	260	228	189	218
Miscellaneous and unclassified	35	37	38	34	36	48	44	47	41	54
Total[d]	1,106	1,169	1,227	1,202	1,299	1,348	1,398	1,451	1,177	1,352

[a] Source: U.S. Bureau of Mines Minerals Yearbooks.

[b] Some of the oxides in this category are used in battery manufacture.

[c] Principal components of this category are gasoline antiknock additives (TEL and TML).

[d] Totals may not equal sum of figures shown because of rounding.

TABLE 4.11. LEAD CONSUMPTION IN THE UNITED STATES, BY PRODUCTS, 1973-1976[a]

	Consumption, Metric Tons			
	1973	1974	1975	1976
Metal Products				
Ammunition	73,901	78,990	68,100	66,664
Bearing metals	14,200	13,250	11,050	11,848
Brass and bronze	20,620	20,170	12,160	14,203
Cable covering	39,005	39,390	20,045	14,448
Caulking lead	18,191	17,900	12,965	11.314
Casting metals	6,550	6,610	6,990	6,084
Collapsible tubes	2,594	2,255	2,010	2,112
Foil	4,521	6,250	4,915	4,649
Pipes, traps, and bends	19,310	14,925	12,910	12,506
Sheet lead	21,218	19,315	22,550	22,165
Solder	65,095	60,115	52,010	57,434
Storage batteries				
Grids, posts	331,560	335,070	296,330	348,146
Oxides	366,328	417,585	338,040	397,773
Terne metal	2,410	2,090	1,370	1,446
Type metal	19,883	18,610	14,700	13,611
Total	1,005,386	1,072,525	876,145	984,403
Pigments				
White lead	1,586	1,810	2,265	2,714
Red lead and litharge	81,228	87,220	59,370	77,460
Pigment colors	15,385	15,725	9,630	15,087
Other[b]	432	650	455	508
Total	98,631	105,405	71,720	95,769
Chemicals				
Antiknocks	248,889	227,205	189,205	217,460
Miscellaneous	856	640	165	133
Total	249,745	227,845	189,370	217,593
Miscellaneous Uses				
Annealing	3,604	3,715	2,385	2,625
Galvanizing	1,174	1,510	1,110	1,135
Lead plating	675	450	340	350
Weights and ballast	18,909	19,425	18,155	20,285
Total	24,362	25,100	25,990	24,395
Other, unclassified uses	19,726	21,855	19,250	29,345
Grand Total[c]	1,397,850	1,452,730	1,178,475	1,351,505

[a] Source: U.S. Bureau of Mines Annual Minerals Yearbooks.

[b] Includes lead content of leaded zinc oxide and other pigments.

[c] Includes lead which went directly from scrap to fabricated products.

posts connected to them and the battery filled with sulfuric acid. The battery is then charged, washed, painted, and shipped. Process steps are shown schematically in Figure 4.6.

Grids, the structural part of the lead plates, are cast by pouring molten lead into the casting machine. Antimonial lead was favored heretofore for this use because of its greater structural strength; as described later it is being challenged by other lead alloys. After removal from the casting machine, the grids are inspected and sent to the pasting operation.

Lead oxide is produced by the fuming process in some battery plants while other plants purchase the oxide. The black oxide (lead oxide containing 20 to 40 percent metallic lead) is mixed with dilute sulfuric acid and various additives such as wood fiber to add strength. The grids are then coated with the oxide paste and flash dried to remove surface water. The plates are then dried under controlled (curing) rates for 2-3 days.

After curing, the plates plus separators are assembled into elements and welded, with lead, to form an electrical connection between the positive and negative plates. These elements are placed in electrolytic baths filled with acid and "formed" by passage of electrical current in the dry-charged battery process. This operation converts the paste to lead peroxide in the positive plate and to sponge lead in the negative plate. The elements are removed from the bath, rinsed with water, and dried. The dried elements are then assembled in a case, the posts welded in and the cover sealed to the case.

Wet-charged storage battery manufacture is somewhat different. The elements are assembled in a case, welded together, the posts connected, and acid added. The batteries are then "formed" by the passage of electrical current as above, and the acid removed for recovery. New acid is added and the battery is boost charged. The final operations are washing and painting.

4.3.1.2 Production and Industry Structure--

The domestic battery industry is dominated by six large corporations operating 65 facilities which produce an estimated 90 percent of all SLI and industrial batteries in the U.S. The remaining 10 percent is distributed among an estimated 110 independent nonintegrated battery manufacturers serving specialized or regional markets. The principal companies and their estimated market shares are shown in Table 4.12. These data are not claimed to be accurate but do roughly approximate the structure of the industry. It is apparent from the data that the industry is an oligopoly containing a fringe of small independents, which, for regional or specialized reasons, can compete with the six national companies which have roughly 90 percent of the total market. The small number of major producers would make easier the implementation of limitations on production or use (Short and Associates, 1976).

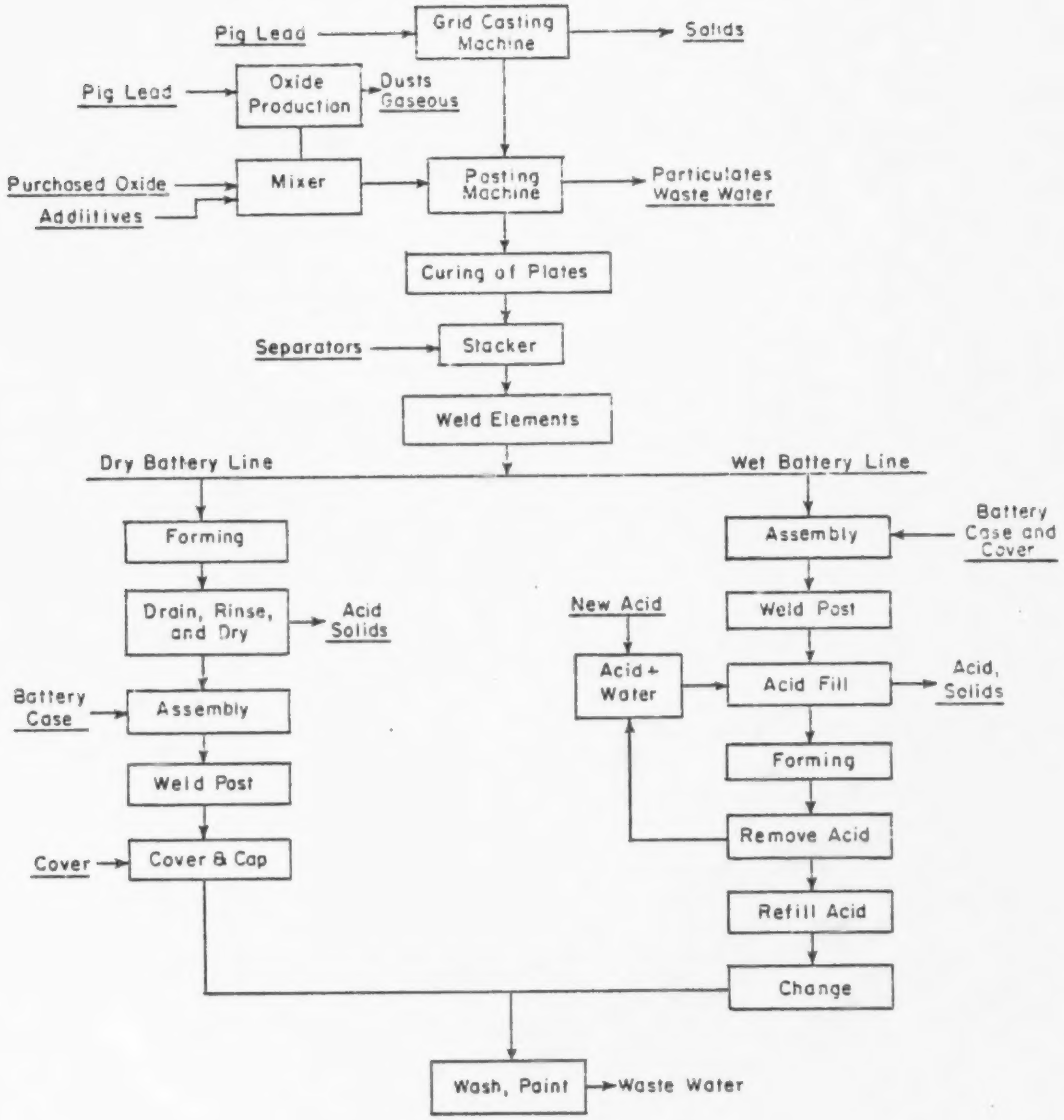


Figure 4.6. Simplified flowsheet for storage battery manufacture.

Source: Crandall and Rodenberg (1974) Reprinted by permission of Purdue University.

TABLE 4.12. ESTIMATED STRUCTURE OF STORAGE BATTERY INDUSTRY[a]

Holding Company	Number of Facilities	Estimated 1974 Production, 10^6 Batteries	Estimated Percent of Market Share	Estimated Number of Employees Exposed to Lead
ESB, Inc.	20	10.00	17.1	3,035
General Battery Company	10	8.94	15.3	2,934
Globe-Union	10	8.96	15.3	2,059
Gould	13	9.60	16.4	2,181
Eltra Corp.	9	7.02	12.0	1,592
Delco-Remy	3	7.98	13.7	1,275
Total large producers	65	52.60	89.8	13,076
Independents	110	5.90	10.1	3,500
Total	175	58.50	100.0	16,575

[a] Source: Adapted from Short and Associates, (1976).

It should be noted that storage batteries constitute a nondissipative use of lead, so that a large fraction of this consumption is recycled secondary lead rather than virgin primary lead. Estimates of extent of recycle of storage batteries are conflicting. One recent industry estimate was that about 50 percent of those scrapped in a given year are reclaimed, via secondary refiners (Anonymous, 1977). Industry sources claim that the recycling of 75 percent of the batteries scrapped would represent a realistic maximum level that probably could be achieved between 1980 and 1985. On the other hand, a Bureau of Mines estimate (Ryan and Hague, 1976) states that about 80 percent of the lead used in the manufacture of storage batteries is recycled.

Thus, battery manufacturing plants, like their supporting secondary refiners, though geographically widely distributed, are concentrated in the more populous urban states, led by California, New York, Pennsylvania, and Illinois.

Paralleling the increase in automobile population, storage batteries have comprised the one growth area of the lead industry (See Table 4.10). Approximately 47.2 million SLI batteries were produced in 1975, 8.5 million less than 1974. Of this total, about 40.7 million were replacement batteries and 6.5 million were original equipment. As shown in Table 4.11, in 1975 the battery grids and posts consumed over 296,000 metric tons of lead, and the oxides for filling the grids consumed over 338,000 metric tons; the two combined represented 53 per cent of total lead consumption in 1975 for all purposes (U.S. Bureau of Mines, 1976).

4.3.1.3 Future Projections--

In the view of Short and Associates (1976) lead storage batteries are technologically and economically superior to any known substitute. Demand for batteries is almost totally a function of automobile production and the number of vehicles in operation, and is, therefore, relatively inelastic. The Bureau of Mines projects a growth in lead demand for batteries to range between 1.08 and 1.9 million metric tons by 2000, with a most probable value of 1.45 million tons, based on the expected rate of growth (about 3.5 percent) of the gross national product (GNP), and evolutionary improvements in lead-acid batteries. The higher value envisions a significant growth in electrically powered vehicles; the lower value is predicated on an accelerated growth of non-lead storage batteries.

A somewhat more optimistic estimate is suggested by Wright and Kubulak (1976) who describe the rate of growth of SLI batteries (each containing about 9 kg of lead) at 4 to 5 percent per year, to which must be added the larger industrial batteries and standby power batteries--markets growing at an estimated 8 to 10 percent per year.

Some major technological and economic changes appear to be in store as a result of the recent development of the "maintenance-free" battery, which will affect not only the lead-acid battery industry, but also the secondary lead industry.

One battery manufacturer recently announced a new heavy-duty battery based on a lead-calcium alloy, impervious to vibration, corrosion-free, and requiring no water make-up. This lead-calcium battery retains its charge 6 to 8 times longer than ordinary batteries and has up to 30 percent more cranking power (U.S. Bureau of Mines, 1975). Competing manufacturers have developed lead alloys containing strontium, or cadmium - antimony in response. This new alloys are expected to virtually replace the conventional antimonial lead heretofore used by all manufacturers. One estimate is that by 1980, 70 percent of all automotive batteries will be made without filler caps (using the new alloys), and conventional antimonial lead's share of the market (61 percent in 1976) will drop to 10 percent. (Walsh, 1977).

This development will have a significant impact upon the secondary lead industry, which up to now has had only one alloy to deal with. In the future scrap battery alloys may contain antimony, calcium, strontium, cadmium, arsenic, tin, aluminum, and possibly other metals (Walker, 1977). This will pose some serious metallurgical problems for secondary refiners. Additionally, there is a very large pool of antimony contained in the present battery population, which will be excess for future battery needs. Needed will be a lead-antimony separation process, and new uses for antimony.

4.3.2 Gasoline Antiknock Additives

The second largest use of lead, and one which is totally dissipative over the entire country, is for the manufacture of gasoline antiknock additives. Lead alkyls, tetraethyl lead (TEL) and tetramethyl lead (TML), provide the least expensive way to raise the octane rating of gasoline, and in recent years all gasolines have contained from 1.5 to 3 g/gal of lead. In general, and for larger refiners, this need for octane improvement resulted in the use of about 2.4 g/gal in the 1960's.

4.3.2.1 Manufacture--

Tetraethyl and tetramethyl lead are produced by two major generic processes--the sodium-lead alloy process and the electrolytic process. Most of these alkyls are produced by the sodium-lead alloy process operated in batch fashion. One major manufacturer operates the sodium-lead alloy process in continuous fashion in two plants and a small manufacturer operates the electrolytic process in one plant. (See Table 4.13) Many of the details of lead alkyl manufacture are proprietary. The general outlines of the processes have been described by Shapiro and Frey (1968) of the Ethyl Corporation, on which the following discussion is partially based. Some supplementary information was obtained in plant visits and discussions with plant personnel.

4.3.2.1.1 Sodium-lead alloy process--A simplified block diagram of the sodium-lead alloy process as applied to tetraethyl lead is presented in Figure 4.7. In this process, the major unit processes are:

TABLE 4.13. LEAD ALKYL MANUFACTURERS AND ESTIMATED CAPACITIES[a]

Thousands of metric tons

	Location	Process	1976 Estimate Production[a]	 Capacity[a]	Total Annual Capacity[b]
E. I. duPont de Nemours & Co., Inc.	Deepwater NJ	Continuous	86	95	
Organic Chemicals Department	Antioch CA	Na-Pb alloy	27	59	154
Petroleum Chemical Division					
Ethyl Corporation	Baton Rouge LA	Batch	91	104	
	Pasadena TX	Na-Pb alloy	27	54	158
Nalco Chemical Company	Freeport LA	Electrolytic			
Petroleum and Process		(TML only)	13.6	18	18
Chemicals Division					
PPG Industries, Inc.	Beaumont TX	Batch			
Chem Division		Na-Pb alloy	41	54	54
Houston Chemical Co.		(TEL only)[c]			
Total			285		385

[a] Source: Battelle - Columbus estimates.

[b] Chem. Marketing Reporter, 209 (26), 9 (June 28, 1976).

[c] TML purchased from Nalco Chemical Company.

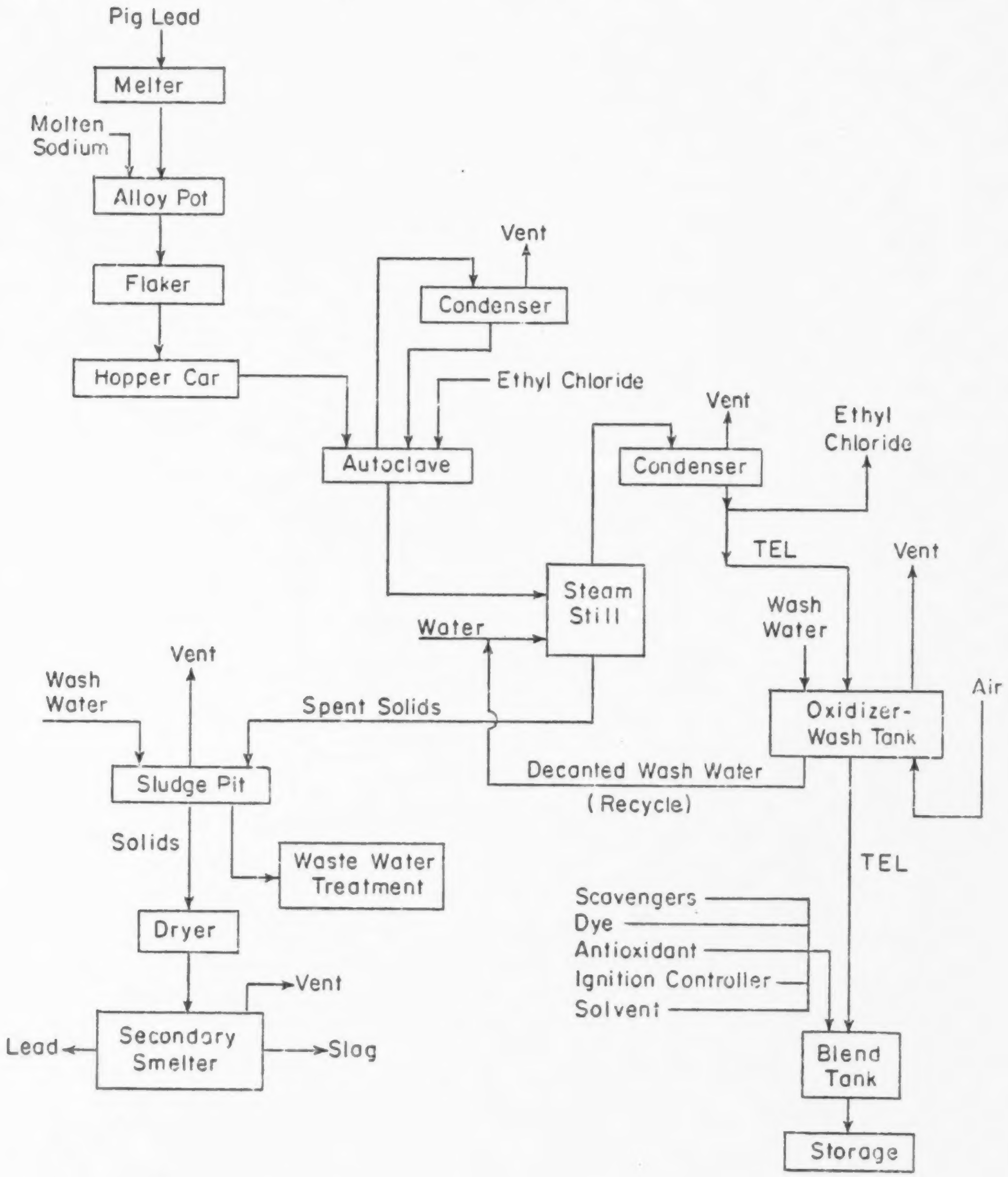


Figure 4.7. Sodium-lead alloy process for the manufacture of tetraethyl lead.

Source: Battelle-Columbus, based on Shapiro and Frey (1968).

- Preparation of the sodium-lead alloy
- Reaction of the alloy with ethyl chloride
- Steam distillation of excess ethyl chloride and tetraethyl lead from a mixture of sodium chloride and lead
- Oxidation and washing
- Lead recovery operations.

The initial step in the process is to melt metallic lead and sodium which are then alloyed in a vessel by combining 90 parts lead with 10 parts sodium. The alloy is then solidified on flakers and conveyed (often in hopper cars) to the autoclaves. Because of the high reactivity of metallic sodium and sodium-lead alloy with oxygen, moisture, and carbon dioxide, all of the steps involving these materials are conducted under a nitrogen atmosphere.

The reaction of ethyl chloride with sodium-lead alloy is performed in autoclaves where temperatures somewhat in excess of 75 C and pressures exceeding about 5 atmospheres pressure (absolute) are employed. The chemical reactions involved are:

$$4Na + 4Pb \longrightarrow 4NaPb$$

$$4NaPb + 4\ C_2H_5Cl \longrightarrow (C_2H_5)_4\ Pb + 4NaCl + 3Pb.$$

Based on these equations, it is apparent that 75 percent of the lead introduced to the autoclave must be recycled.

If tetramethyl lead (TML) is to be produced, methyl chloride is used instead of ethyl chloride. In addition, a Lewis acid such as aluminum chloride or triethyl aluminum and a solvent such as toluene are autoclaved at more severe temperatures and higher pressures.

These reactions are exothermic and significant cooling is provided by condensing volatilized ethyl chloride (or methyl chloride--toluene) and returning the liquid to the autoclave. In the case of TEL manufacture, significant amounts (about 10 percent of theoretical) of ethyl chloride are lost by side reactions to yield ethane, ethylene, and butane which are ultimately vented to an incinerator.

After the reaction is complete, most of the reaction mass is dumped to a steam still, after the autoclave has been depressurized through a condenser to recover a portion of the ethyl chloride (or methyl chloride and toluene). The steam stills are typically operated in batch fashion but may be operated continuously. In the batch distillation, the low boiling ethyl chloride (or methyl chloride and toluene) is the first material distilled; this is condensed and is recycled after purification. TEL (or TML) mixed with steam is volatilized next and is condensed and forwarded to an oxidation-wash operation. The solids, consisting principally of lead and sodium chloride, are discharged to a sludge pit.

The TEL-water mixture is oxidized by sparging air into the mixture, primarily to oxidize and precipitate unwanted metal compounds such as bismuth. (This step is not used with TML manufacture). After oxidation, the TEL (or TML) is washed with water, decanted, and forwarded to the blending operation where scavengers (ethylene dichloride and ethylene dibromide), dye, antioxidant, solvent (kerosene), and a surface ignition control agent, are added to produce the finished antiknock gasoline additive.

The solids which were discharged from the steam still to the sludge pit are washed (or leached) with water to dissolve out the sodium chloride in a covered sludge pot. Air from the covered pot is vented through a carbon bed and subsequently to a stack. Water solution containing the dissolved salt along with fine lead particles and residual TEL constitutes a significant aqueous lead emission and, therefore, receives careful attention from antiknock producers as a waste disposal problem. Treatment may be by proprietary processes which inlude pH adjustment to produce relatively insoluble basic lead carbonate and lagooning, followed by treatment of the effluent with activated carbon. The residual solids are dried in an enclosed dryer so that the volatiles, consisting primarily of water and ethyl chloride, may be condensed. The dried solids are then refined in a conventional secondary smelting operation to recover the lead.

4.3.2.1.2 Electrolytic process--A simplified block diagram of the electrolytic process as applied to tetramethyl lead is presented as Figure 4.8. In this process, the major steps are:

- Preparation of Grignard reagent (methyl magnesium chloride etherate in ether)
- Electrolysis of the Grignard reagent using lead as the anode to produce tetramethyl lead and magnesium chloride
- Recovery of the methyl chloride and ether for recycle and purification of tetramethyl lead from magnesium chloride
- Blending to produce finished antiknock compounds.

Grignard reagent is produced by reacting finely divided metallic magnesium with methyl chloride in the presence of ether which forms a methyl magnesium chloride etherate in ether solution. This solution is introduced to an electrolytic cell which uses lead pellets as the anode and the walls of the cell as the cathode. The overall electrochemical reaction is as follows:

$$2CH_3MgCl + 2CH_3Cl + Pb \longrightarrow (CH_3)_4Pb + 2MgCl_2 \quad .$$

Excess methyl chloride is removed by distillation. The specific process employed to separate TML, ether, and magnesium chloride is not known but probably uses distillation for recovery of the ether and a proprietary solvent extraction process combined with distillation to separate magnesium chloride from TML and to purify the TML.

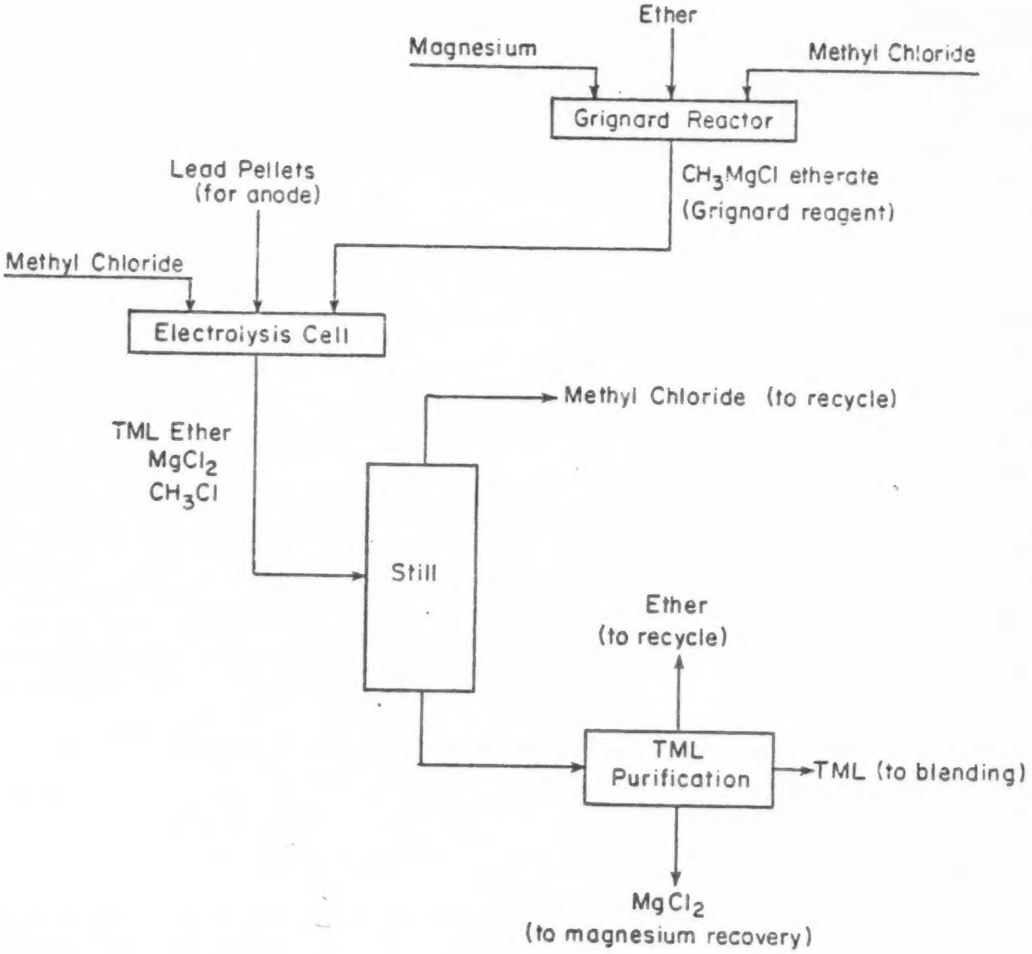


Figure 4.8. Electrolysis process for the manufacture of tetramethyl lead.

Source: Battelle-Columbus, based on Shapiro and Frey (1968).

4.3.2.1.3 Mixed TEL-TML antiknocks--TML and TML-TEL mixtures are most applicable to high octane gasolines containing high aromatics concentrations. Two types of TEL-TML mixtures are produced--simple physical mixtures and reacted mixtures. Preparation of the reacted mixtures involves adding TML to TEL in the presence of a catalyst to yield a mixture of triethyl (mono) methyl lead, diethyl dimethyl lead, and (mono) ethyl trimethyl lead plus unreacted TEL and TML.

4.3.2.1.4 Environmental management practices--The manufacturers are very aware of the high toxicity of the lead alkyls and generally discharge vented gases containing TML and TEL to high stacks or incinerators. When possible, tanks and vessels being filled with liquids containing alkyls are vented back to the tanks being emptied.

The industry has evidently unofficially adopted a 100 ug/m^3 lead level, as proposed by the Department of Labor (1975) for inorganic lead compounds.* Blood and urine samples are regularly taken from employees and monitored to assure that individuals do not become overly exposed.

A significant liquid stream containing lead alkyl is generated from the washing operation after the oxidation step. This stream is recycled to the steam still to recover valuable TEL or TML. A particularly troublesome waste stream is generated during the water wash (or leach) of the salt from the material dumped to the sludge pit. This waste would contain residual TEL or TML plus metallic lead. This waste stream is handled differently by the manaufacturers, by proprietary processes in some cases. One company first applies a treatment to adjust pH and subsequently forms insoluble basic lead carbonate. Next the waste is lagooned followed by passage of the supernate through activated carbon. According to a company representative the final discharge is about 5 pounds per day of lead--about 1 ton per year.

The principal solids generated by the plant are the dried solids from the sludge pit. Lead from this material is recovered by in-plant and in some cases off-site secondary recovery smelting operations in reverberatory furnaces. Slag from this operation receives some treatment and particulates are collected by either baghouses or in venturi scrubbers.

4.3.2.1.5 Transportation and handling--Lead alkyls are recognized as hazardous substances requiring extreme care in transportation and handling.

Nearly all antiknock gasoline additives are transported in the United States by specially designed 3000- to 9600-gallon railroad tank cars or 1000- to 3000-gallon over-the-road tank trucks approved by the I.C.C. However, 775- to 3000-gallon portable tanks and 55-gallon drums are often employed in export shipments; such tanks are approved by the Coast Guard (E. I. DuPont de Nemours and Company, 1964; industry sources).

Because of the toxicity of antiknock gasoline additives the producers have been vitally concerned with the proper handling of such products since their introduction to the market. As a consequence, these producers require that refineries install blending facilities that have been approved at the design and final inspection stages by representatives of alkyl manufacturers. The principal feature of the blending plants is that they operate under vacuum to avoid spills. If spills or leaks do occur in the systems, repairs and corrective measures must be conducted with the advice or personal attendance of the alkyl manufacturer's factory representative. For example, pipelines or tanks that have been used to handle concentrated lead alkyls may not be opened for repair unless a factory representative is in attendance (E. I. DuPont de Nemours and Company, 1964). The manufacturers of lead alkyls control adherence to such rules by simply refusing to ship their product.

---

* Final standard promulgated (U.S. Department of Labor, 1978) is 50 ug/m^3.

The manufacturers of lead alkyls have published 10- to 15-page books of "regulations" or "guides" which were based on the advice of a committee appointed by the Surgeon General of the U.S. Public Health Service. (E. I. DuPont de Nemours and Company, 1964; Ethyl Corporation, undated). Matters regulated include selection of blending personnel, emergency procedures, tank car and drum unloading procedures, cleanup of spills, laboratory procedures, precautions for men entering tanks that have contained antiknocks, required clothing and equipment, bathing procedures, required posted notices, and many other important aspects. These regulations or guides generally appear to be those of the manufacturer. However, analysis of the regulations of the two largest lead alkyl manufacturers reveal many similarities; therefore,it is believed that the committee appointed by the Surgeon General required many specifics to be incorporated.

The guidelines emphasize that concentrated lead antiknocks should under no circumstances be heated in the laboratory or distilled,, even under vacuum, because of the danger of explosion.

4.3.2.2 Production and Industry Structure--

There are four manufacturers of TEL and TML as shown in Table 4.13. DuPont and Ethyl Corporation dominate the industry, each possessing about 40 percent of the business, with Houston Chemical and Nalco sharing the remainder. Industry capacity is estimated at 1.35 billion to 1.45 billion lb of finished antiknock mix (Anderson, 1978), which translates to 375,000 to 395,000 metric tons/yr of 100 percent alkyl. At the present time the industry has significant over-capacity relative to current demands; as discussed in the following section, this situation will inevitably become worse as the lead phasedown proceeds.

It is estimated that in 1976 about 277,000 metric tons (305,000 short tons) of lead alkyls were produced, and thus, the industry was operating at about 75 percent capacity. Because of the special environmental situation in California, it is believed that DuPont's Antioch, California plant is operating at a disproportionately lower rate than the other plants. In addition, it is believed that Ethyl's Pasadena (Houston) plant is operating at a lower operating rate than its Baton Rouge plant.

Alkyl lead production, sales, and domestic consumption for the period 1966-1976 are presented in Table 4.14. The apparent discrepancy between production and sales is principally due to double counting, i.e., the manufacturers may report production of tetraethyl lead when produced and again when such TEL is used to produce mixed TEL and TML. A large number of antiknock additives are sold. In addition to TEL and TML, mixtures containing triethyl methyl lead, diethyl dimethyl lead, or ethyl trimethyl lead are also manufactured. Value of annual sales in recent years has been in the 500 to 600 million dollar range.

The high compression engines of the early 1960's required high-octane gasoline, and the average lead content of the gasoline pool peaked in the late 1960's (Table 4.15 and Figure 4.9). Since about the 1970 model year, automobiles have been built with lower compression engines--ones requiring lower octane gasoline and thus gasoline with lower lead content. As a result of this change, practically all new cars built since 1970 are able to use regular gasoline instead of highly leaded premium fuels. (Faoro and McMullen, 1977). The percentage of cars on the road requiring premium gasoline is now down to about 6.5 percent, as illustrated by Figure 4.10 (National Petroleum News, 1978). The result of the engine modifications can be clearly seen in the lower lead content in gasoline (in both regular and premium grades) after 1969.

Thus, the average lead content of the total gasoline pool has also been in a steady decline since 1969, when it peaked at 2.53 g/gal. In 1975 the average lead content of the pool was for the first time below that of regular gasoline; by 1978 the estimated average lead content of the pool was 1.20 g/gal. If the EPA phase-down schedule is met, this will further decrease to 0.59 g/gal after October 1, 1979.

Historical consumption data for total gasoline and lead are shown in Table 4.16. Gasoline consumption data are based on Ethyl Corporation surveys, adjusted to compensate for the approximately 95 per cent coverage of refiners. Average pool lead contents are based on the semi-annual surveys of the Bartlesville Energy Technology Center. Lead consumption peaked in 1970; the peak in gasoline consumption does not appear to have yet been reached. Lead consumption so calculated does not agree with consumption data reported by the U.S. Bureau of Mines. For example, lead consumed in 1975 for antiknock additive manufacture, according to the U.S. Bureau of Mines data (Table 4.11), was 189,200 metric tons. However, according to the accounting system used this includes all lead sold to antiknock additive manufacturers for the manufacture of lead alkyls. Thus, lead for alkyls destined for export (40 to 50 thousand tons annually in recent years) is included in this figure (U.S. Bureau of Mines, 1978) Also, utilization of the lead is not 100 percent, and some is presumed to be sufficiently degraded to drosses, etc., during alkyl manufacture such that it is recycled to the secondary lead-segment of the industry. The 1975 estimate of 162,800 metric tons, based on actual lead content of gasolines, is believed to be a better estimate and is used for purpose of calculating emissions.

TABLE 4.14. PRODUCTION AND CONSUMPTION OF LEAD ALKYLS, 1966-1976[a]

Thousands of metric tons

Year	TEL[a]	TML[a]	TML/TEL Mixtures[a,b]	Exports and Consumption[c]	U.S. Consumption
1966	247	50	--	311	280
1967	251	43	92	315	283
1968	220	53	139	327	298
1969	168	35	195	336	313
1970	147	--	229	342	312
1971	128	--	--	327	297
1972	137	--	304	337	305
1973	161	--	347	335	299
1974	210	--	207	317	262
1975	142	--	160	277	238
1976	--	--	--	277[d]	238[d]

[a] Source: Synthetic Organic Chemicals, U.S. Production and Sales, U.S. International Trade Commission (formerly Tariff Commission) (1977.)

[b] Includes tetramethyl lead, mixtures of TML and TEL, and other organic lead compounds.

[c] Source: Chemical Economics Handbook, Stanford Research Institute (1976), except as noted.

[d] Battelle-Columbus estimates.

TABLE 4.15. AVERAGE LEAD CONTENT OF GASOLINES, 1965-1978

Year	Average Lead Content By Grade, g/gal[a]		Percentage of Total Automotive Gasoline Sales[b]			Average Lead Content in Gasoline Pool, g/gal
	Leaded Premium	Leaded Regular	Leaded Premium	Leaded Regular	Unleaded	
1965	2.76	2.11	37.2	62.7	-	2.35
1966	2.81	2.19	39.0	60.9	-	2.43
1967	2.81	2.27	39.7	60.2	-	2.49
1968	2.79	2.28	41.2	58.7	-	2.49
1969	2.80	2.34	41.9	58.0	-	2.53
1970	2.73	2.28	42.6	57.4	-	2.47
1971	2.59	2.09	41.1	58.9	-	2.29
1972	2.45	1.94	38.1	61.9	-	2.13
1973	2.34	1.87	32.4	67.6	-	2.02
1974	2.23	1.76	24.5	75.5	-	1.88
1975	2.19	1.73	19.0	68.0	13.0	1.60
1976	2.37	1.85	17.1	62.7	20.2	1.57
1977	2.36	1.82	14.8	58.6	26.6	1.42
1978	2.70[c]	1.68[c]	12.3[d]	54.4[d]	33.3[d]	1.20[d]

[a] Data from Shelton, E.M., Bartlesville Energy Research Center.

[b] Data from yearly Report of Gasoline Sales, By States, 1977, compiled by Ethyl Corporation.

[c] Battelle-Columbus estimate based on partial 1978 data.

[d] Battelle-Columbus estimates.

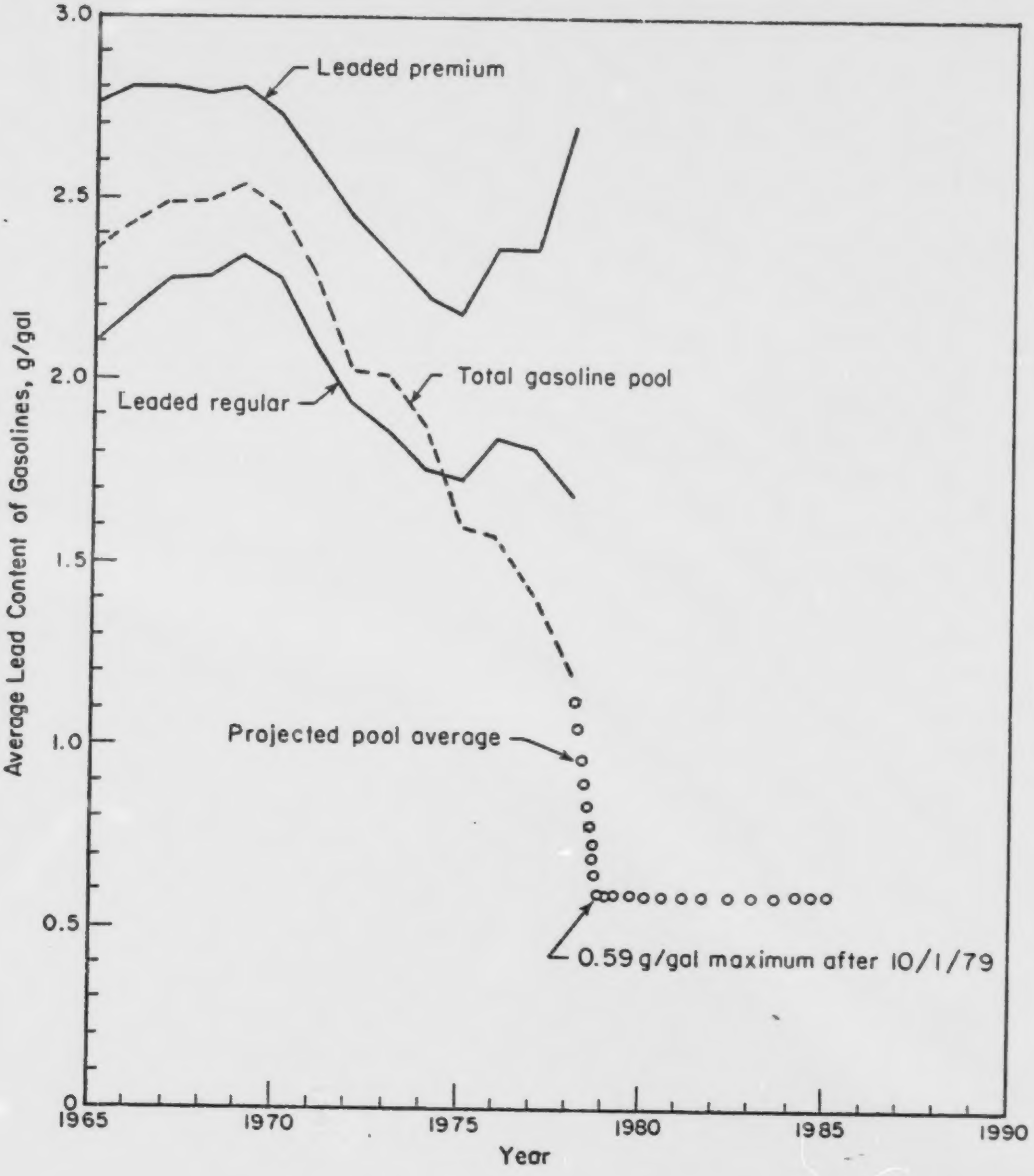


Figure 4.9. Nationwide trends in gasoline lead content, 1965-1978.

Source: Shelton, E.M.

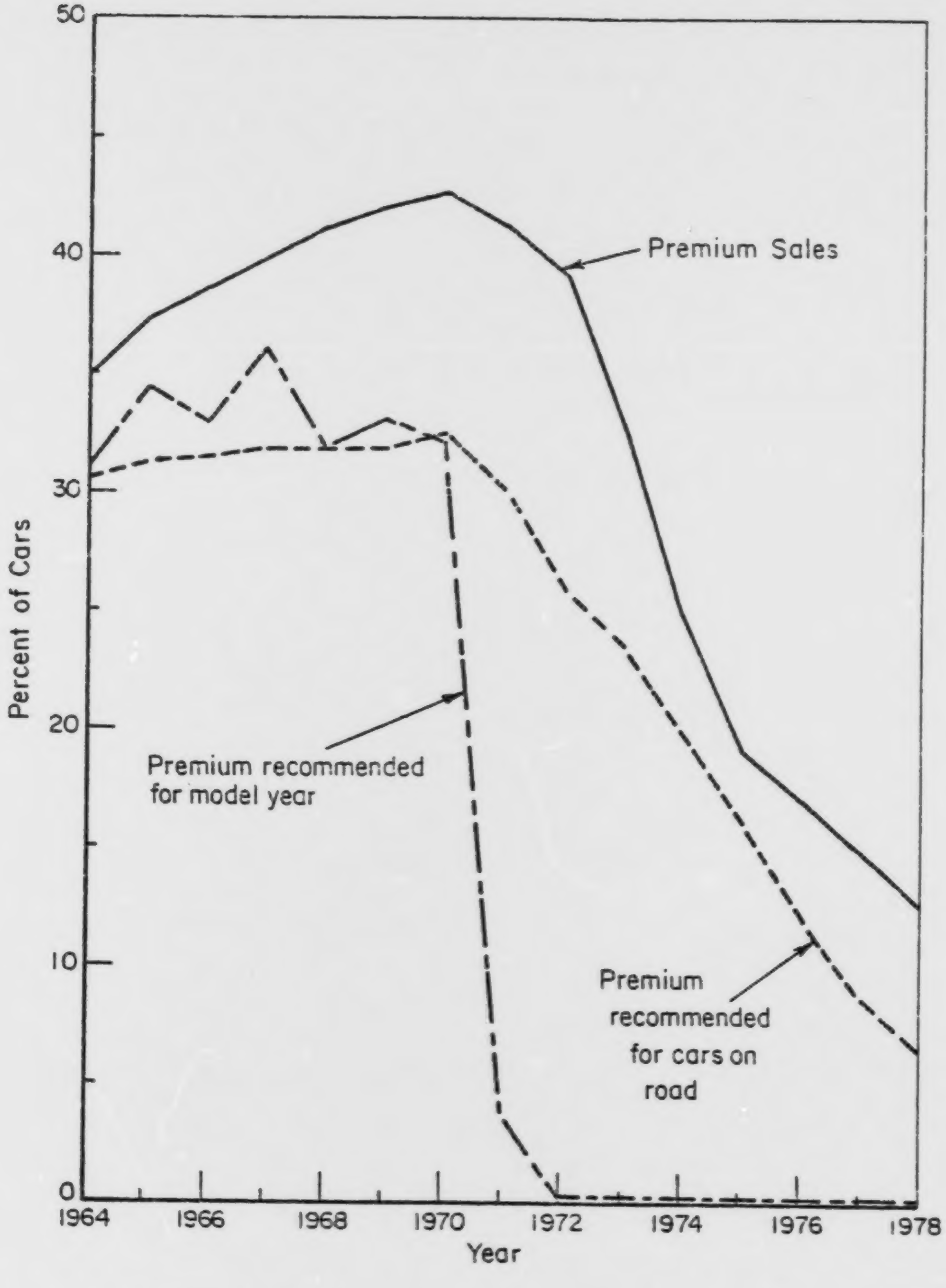


Figure 4.10. Passenger car use of premium gasoline
Source: National Petroleum News (1978).

TABLE 4.16. CONSUMPTION OF GASOLINE AND LEAD ANTI-KNOCK, 1955-1978

Year	Total Gasoline Sales, Billion Gal[a]	Average Pool Lead, g/gal[b]	Lead, Metric Tons/Yr
1955	51.8	2.38	123,300
1956	54.0	2.44	131,800
1957	55.5	2.38	132,100
1958	56.9	2.14	121,800
1959	59.8	2.12	126,800
1960	61.0	2.04	124,400
1961	61.8	1.94	119,900
1962	63.9	2.01	128,500
1963	66.2	2.15	142,300
1964	69.1	2.25	155,500
1965	71.7	2.35	168,500
1966	74.9	2.43	181,900
1967	77.2	2.49	192,200
1968	81.6	2.49	203,200
1969	85.3	2.53	215,900
1970	89.1	2.47	220,200
1971	92.9	2.29	212,700
1972	97.3	2.13	207,200
1973	101.8	2.02	205,600
1974	99.2	1.88	186,500
1975	101.8	1.60	162,800
1976	106.9	1.57	167,800
1977	109.1	1.42	154,900
1978	112.8[c]	1.20[c]	135,400

---

[a] Based on Ethyl Corporation data, increased 4 per cent to adjust for 95-96 percent industry coverage of survey.

[b] Based on Bartlesville Energy Technology Center semi-annual data.

[c] Estimated.

4.3.2.3 Future Projections--

In addition to promulgating regulations to control hydrocarbon emissions, which led to the adoption of catalytic converters as the preferred abatement measure (and consequently, to no-lead gasoline for vehicles so equipped), the U.S. Environmental Protection Agency also promulgated regulations reducing the average lead content of all gasoline sold to 0.5 g/gal by October 1,1979 (U. S. Environmental Protection Agency, 1973; 1976). This has had and is having a major impact upon the petroleum refining industry as well as upon the average lead content of gasolines now and in the future.

The use of lead for gasoline antiknock additivives has already begun to decline, and the decline will accelerate as the more drastic steps of the lead phase down are implemented. Initially, the average lead content of the total pool was to have been reduced to 0.8 g/gal by January 1, 1978. However, this proved to be an overly ambitious goal, and the EPA Administrator was empowered to grant waivers to refiners exhibiting "good faith" efforts to reach the 0.8 g/gal goal. Most refiners qualified for such waivers, which accounts for the 1.2 g/gal pool average for 1978. What the average lead content of the total gasoline pool will be by 1980 and beyond is a matter of some speculation, compounded by numerous and ill-defined factors. A level not exceeding 0.5 g/gal is mandated by EPA regulations.* It may be lower than this. One uncertain factor is how much gasoline will be consumed in future years. Total car mileage is projected to increase, more or less proportionately to population growth and long-term secular trends. However, gasoline demands are less certain, as is the fraction of that demand represented by pre-1974 cars and light trucks able to utilize leaded gasoline.

Significant improvements in average automobile fuel economy have been mandated by EPA. Also, it is well known that older cars are driven fewer miles per year than new cars. For example, a typical 10-year-old car is driven half as many miles per year as the "average" car, and one-third the number of miles per year as the car less than one year old (Sobotka, 1976). On the basis of this and similar considerations, Sobotka estimated the following projection of motor gasoline grade distribution:

	Percent of Volume		
		Leaded	
Year	Unleaded	Regular	Premium
1976	20	64.5	15.5
1980	56	37	7
1985	77	21	2
1990	87	13	--

---

* Although the originally promulgated maximum lead content for total pool was 0.50 g/gal, exemptions for small refiners have raised the national target limit to approximately 0.59 g/gal.

Similar estimates are projected by the "ESCON" model developed by one of the major TEL manufacturers (E.I. DuPont de Nemours and Co., Inc., 1978). Estimates of total gasoline consumption through 1990 are summarized in Table 4.17, and illustrated by Figure 4.11. It will be noted that according to this forecast, total gasoline consumption is predicted to peak in 1980-81, and decline slightly thereafter. This forecast is consistently below the one made in 1977, resulting from a lower forecast of motor vehicle sales, and a more conservative projection of vehicle use. The model assumes that automobile manufacturers will meet Federal targets on fuel economy.

The effect of emission control regulations will be that by 1980 unleaded gasoline will have captured half the total market, and its share will continue to increase, reaching 78 to 80 percent of the total by 1985 and 85 percent by 1990. This will permit increasing lead levels in the decreasing volume of leaded gasoline while still maintaining the required 0.5 g/gal average for the total pool. Anderson (1978) estimates that by 1985 the lead content of leaded gas will be back up to what it was in 1971 (Figure 4.12).

Another factor which will increase unleaded gasoline's share of the market was the shift, beginning with the 1979 model year, in maximum weight rating for light duty vehicles from 6,000 lbs to 8,500 lbs, gross vehicle weight (GVW). Previously, EPA exempted trucks over 6,000 lbs from emission control, and as Prescott (1976) pointed out, even most full-size pickup trucks weigh more than this amount. Since light trucks account for nearly 20 percent of gasoline consumed and about half fall in the 6,000 to 8,500 lb weight bracket, a significant use of leaded gasoline is being eliminated.

The possibility of an "octane crunch" about 1980-1981 has been suggested, especially if gasoline demand exceeds forecasts, and there is a shortfall in supply of high-octane gasoline feedstocks. Octane quality of all three grades of gasoline appears to have been reasonably satisfactory through 1978. As indicated by Table 4.15, market share of leaded premium gasoline peaked in 1970-71 at about 42.6 percent. As lower compression engines were adopted in succeeding model years, the need for leaded premium gasoline decreased; by 1978 it retained only about a 12 percent market share (Figure 4.10). The lessened need for high-octane premium gasoline permitted refiners to achieve desired octane levels by increasing the content of high octane components and reducing the rate of lead addition during the 1970-75 period. As the demand for unleaded gasoline increased beginning in 1975, it became necessary to divert more of the high-octane feedstocks from premium to unleaded gasoline. The resulting octane deficiency in leaded premium gasoline has been made up by increasing its lead content, as illustrated by lead levels for the last several years (Figure 4.9). However, utilization of this strategy will lose much of its effectiveness after October 1, 1979, when the lead content of the total pool must be reduced to the 0.59 g/gal maximum limit. After that date the possibility of an "octane crunch" increases significantly. This is perhaps best illustrated by developing a scenario and calculating the probable outcome.

TABLE 4.17. GASOLINE CONSUMPTION FORECAST[a]

	Total Gasoline Use, All Vehicles						
	Billion Gallons per year				Percentage of Sales		
Year	Leaded Premium	Leaded Regular	Unleaded	Total	Leaded Premium	Leaded Regular	Unleaded
1979	11.2	55.1	48.3	114.5	9.8	48.1	42.1
1980	8.7	48.6	57.8	115.0	7.5	42.2	50.3
1981	6.5	42.1	66.1	114.7	5.7	36.7	57.5
1982	4.8	36.4	71.3	112.5	4.3	32.4	63.3
1983	3.5	31.4	75.2	110.1	3.2	28.5	68.3
1984	2.6	27.3	78.3	108.2	2.4	25.2	72.4
1985	1.9	24.1	80.9	106.9	1.7	22.5	75.7
1986	1.3	21.4	82.2	104.9	1.2	20.4	78.3
1987	0.8	19.2	82.9	103.0	0.8	18.6	80.5
1988	0.5	17.4	83.5	101.4	0.5	17.1	82.3
1989	0.3	15.8	83.8	100.0	0.3	15.8	83.8
1990	0.2	14.5	84.1	98.8	0.2	14.7	85.1

[a] Source: Data from ESCON model (E. I. DuPont de Nemours and Co. Inc., 1978)

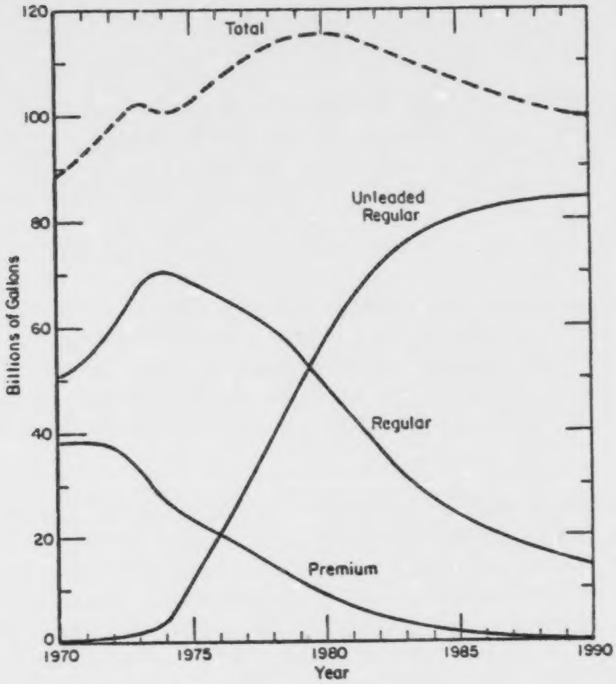


Figure 4.11. Projected consumption of grades of gasoline, 1970-1990
Source: E.I. DuPont de Nemours (1978).

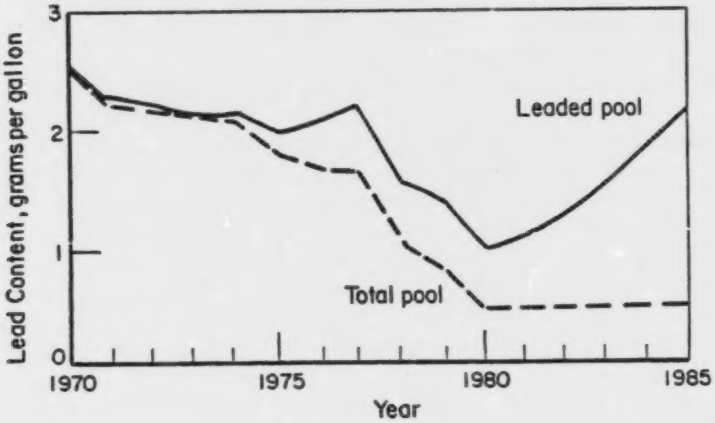


Figure 4.12. Estimated lead content of leaded and total gasoline pool, 1970-1985.

Source: Anderson (1978). Reprinted with permission from Chemical and Engineering News. (c) American Chemical Society (1978).

Principal assumptions of the proposed scenario are that quantities and grades of gasoline are as projected by the ESCON model (Table 4.17); that without phasedown the refining industry can achieve the slight increase in clear pool octane required to maintain quality; and that leaded regular and premium gasolines contain 2.0 and 2.5 g/gal, respectively. As illustrated by Table 4.15, these values are representative of recent ranges.

Results of the analysis are summarized in Table 4.18. Assuming that octane levels are maintained, this estimate suggests that the critical years may be 1980-81 when octane improvement of the leaded gasoline pool will be needed which is equivalent to that which, under normal industry practice, would be achieved by an additional 30,000 to 50,000 metric tons of lead.

An octane deficiency may be overcome by increasing industry capacity for catalytic reforming, catalytic cracking, alkylation, and isomerization to boost the octane level of the clear pool. Whether this increase can or will occur by 1980 is problematical. Alternatively, some octane deficiency may be met by some lowering of average octane levels. This cannot all come from the leaded pool; a proportionate share must come from the unleaded pool. However, if engine performance suffers as a result, there will be an increased tendency towards "misfueling" i.e., the use of leaded gasoline in cars equipped with catalytic converters, promoting catalyst poisoning, and a consequent increase in emissions of hydrocarbons.

If the phasedown proceeds as planned, lead consumption will be reduced to about 67,850 metric tons per year in 1980, and remain below this level thereafter (Table 4.18). Interestingly, beginning in 1984, lead emissions would be lower with the assumed lead contents of 2.0 and 2.5 g/gal for regular and premium gasoline than with a 0.59 g/gal limit for the total pool. At these lead levels, average lead concentrations in the total pool would be 0.50 g/gal in 1985, and only 0.30 g/gal by 1990.

The lead consumption data reported has been that used for domestic gasoline supplies. Exports of lead alkyls have been at about the 40,000 tons/yr level and are estimated to continue at that level. Exports are expected by some industry representatives to grow significantly in the future years. However, recent European actions indicate that decreases in lead contents of gasoline will be comparable to those in the U.S. Prescott (1976) reports that the latest European Economic Community objective is to reduce lead in premium gasoline to 0.4 g/l (1.5 g/gal) and in regular to 0.15 g/l (0.6 g/gal) by January 1, 1978. West Germany has been at 0.6 g/gal since the beginning of 1976. These and other similar developments suggest that the European lead alkyl export market can also expect some shrinkage; however, it is also estimated that increases in markets in Latin America and other areas will offset this decrease.

Because of the projected decline in lead alkyl production in the United States, it is quite improbable that any new production facilities will be constructed. In fact, it is believed that DuPont's Antioch, California

TABLE 4.18. EFFECT OF PHASEDOWN ON CONSUMPTION OF LEAD IN GASOLINE

		Lead, metric tons/yr			
			Lead Required for Leaded Gasoline at "Typical" Levels		
Year	Total Gasoline, Billion Gal.	At 0.59 g/gal in Pool	Premium	Regular	Total
1979	114.5	119,940[b]	28,000	110,200	138,200
1980	115.0	67,850	21,750	97,200	118,950
1981	114.7	67,670	16,25[illegible]	84,200	100,450
1982	112.5	66,380	12,000	72,000	84,000
1983	110.1	64,960	81,750	62,800	71,550
1984	108.2	63,840	6,500	54,600	61,110
1985	106.9	63,070	4,750	48,200	52,950[c]
1986	104.9	61,890	3,250	42,800	46,050
1987	103.0	60,770	2,000	38,400	40,400
1988	101.4	59,830	1,250	34,800	36,050
1989	100.0	59,000	750	31,600	32,350
1990	98.8	58,290	500	29,000	29,500[d]

[a] Based on projected gasoline consumption (Table 4.17) and 2.00 g/gal lead in regular and 2.50 g /gal in premium (Table 4.15).

[b] Assumes 1978 pool average of 1.20 g/gal for first 3/4 year.

[c] Average of pool = 0.50 g/gal

[d] Average of pool = 0.30 g/gal

plant will be closed in about 1979. In addition, other smaller plants including Ethyl's Pasadena, Texas and Nalco's Freeport, Texas plant may close shortly thereafter. The development of new products and processes is unlikely except that PPG Industries may produce more TML at Beaumont, Texas if Nalco's plant is closed.

4.3.3 Lead Oxides and Pigments

Lead oxides and pigments is a conventional industry grouping in which there are some overlaps. Black oxide is used for batteries and never as a pigment. Litharge (PbO), formerly used as a pigment, is very seldom so used now, but is the most important starting lead compound for the manufacture of other lead compounds. Red lead (Pb_3O_4), another oxide, is still widely used as a primer coating for steel.

4.3.3.1 Manufacture--

4.3.3.1.1 Litharge--Litharge or lead monoxide contains about 93 percent lead. Its color ranges from yellow to reddish yellow, depending on its crystalline structure. (U.S. Tariff Commission, 1968) While it can be used as a pigment, it is used primarily in the manufacture of products other than paint pigments, e.g., batteries, ceramics, glazes, lead glasses, vitreous enamels, and is a convenient and chemically reactive starting material for a wide variety of other lead compounds.

There are several principal processes for producing litharge, all involving the oxidation of elemental lead at elevated temperature. Molten lead may be held in a cupelling furnace at about 1020 C until oxidized to molten litharge, or molten lead at about 510 C may be atomized into a flame, where it burns vigorously, forming "sublimed" or "fumed" litharge. Aternatively, molten lead is fumed from the surface and collected in a baghouse. In all of the processes, the product must be cooled quickly to below about 300 C to avoid further oxidation, forming red lead. (Beltz, et al., 1973)

4.3.3.1.2 Black oxide--Black oxide is a mixture of lead monoxide and finely divided elemental lead, which is used in the manufacture of lead-acid storage batteries. It can be made in the same furnace as used for litharge manufacture; the desired ratio of lead oxide to metallic lead is controlled by the temperature of the charge and the amount of air swept through the furnace. The product is caught in a baghouse, and may be further milled before use.

4.3.3.1.3 Red lead--Red lead, or minium (also known as orange mineral), contains about 91 percent lead by weight. Its color ranges from bright red to orange-red depending on the true red lead content. Its major use is in protective coatings to prevent rusting and corrosion of steel products; the bright color of a prime coat of red lead is a familiar sight on new bridges and other steel structures. It is also used in battery manufacture, and some is used in the manufacture of ceramics.

Red lead is made by further oxidation of litharge, sometimes in the same furnace.

4.3.3.1.4 Leaded zinc oxides--Leaded zinc oxides find some use as white pigments in oil-based exterior paints. They can be prepared by confuming zinc and lead sulfide ores or by physical mixing of lead and zinc oxides. They have largely been replaced by other pigments, and are now almost obsolete pigments. Production data are no longer published in Bureau of Mines tabulations, to avoid disclosing individual company data.

4.3.3.1.5 White lead--The commercial varieties of white lead include basic lead carbonate, basic lead sulfate, and basic lead silicate. As illustrated by the schematic flow sheet in Figure 4.13, basic lead carbonate is made by a wet process involving the reaction of lead with dilute acetic acid and then precipitation with CO_2 as the basic carbonate. The acetic acid liberated by the CO_2 reaction is free to digest more lead. The process is generally carried out as a batch operation. In the older methods, the CO_2 was generated by fermentation; in the newer processes, combustion products are the source of CO_2. (Beltz, et al., 1973).

As illustrated by Figure 4.13, basic lead sulfate and basic lead silicate are generally produced by thermal processes. Lead sulfate fume is produced by spraying molten lead into a combustion chamber along with SO_2. The high temperature causes volatilization and oxidation of the lead. The resulting oxide then reacts with the SO_2 and excess air to yield a basic lead sulfate fume. The fume and flue gas are passed directly to a baghouse filter for collection, and the product is suitable for direct use as the pigment. Basic lead silicate is produced by fusion of litharge (PbO) with silica. This process is carried out at about 1000 C (1800 F). When the fusion is complete, the melt is drained from the furnace into water which serves to quench and granulate the product. The resulting lead silicate is then ready for final finishing by grinding to form the pigment (Battelle-Columbus, 1975).

White-lead paints were for years the traditional exterior house paints for wood (and were used for interior paints as well). This is, of course, a totally dissipative use, although the rate of dissipation to the environment is quite speculative. The quantity of lead that has been applied to structures in the form of paint is truly enormous. Ewing and Pearson (1974) quote the estimate of Ziegfeld (1964) that in the preceding 40 years over 3 million tons of lead paint had been applied, of which a significant fraction was white lead paint on houses.

White-lead pigment is subject to discoloration from sulfur in the atmosphere, and it has been largely replaced by zinc oxide and titanium dioxide but small quantities still are produced annually. (See Table 4.11). However, with the prohibition of the lead content in paint for dwellings exceeding, first 1 percent, then 0.5 percent, and finally now 0.06 percent, as described in a later section, white lead is almost an obsolete pigment.

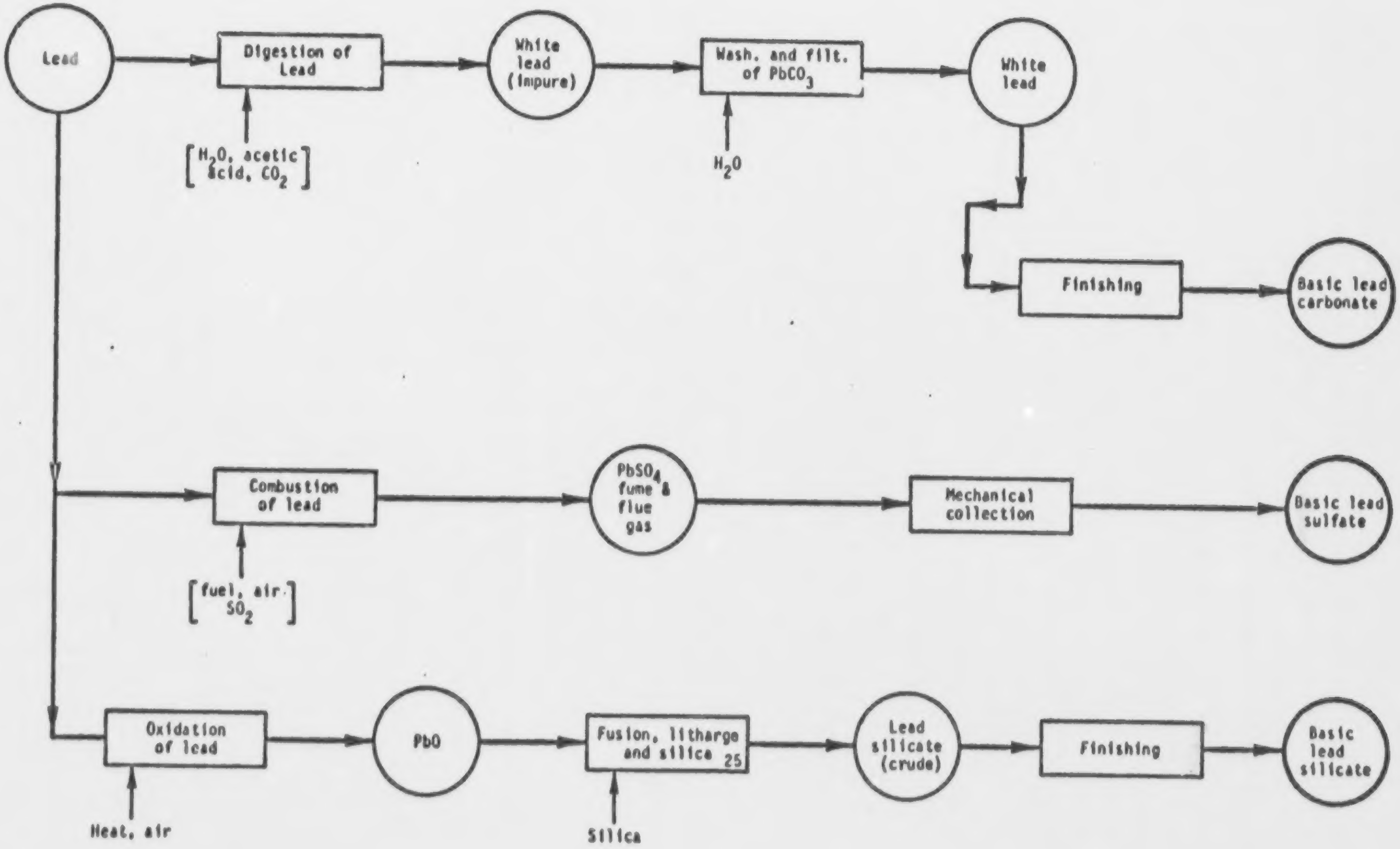


Figure 4.13. Simplified flow sheet of processes for the manufacture of lead-based white pigments.

Source: Battelle-Columbus (1975).

Its consumption has fallen from 32,800 metric tons in 1950 to 2,700 tons in 1975, a decrease of 92 percent, and it can be expected to decline still further.

4.3.3.1.6 Lead chromate-based pigments--Lead-based pigment colors include yellows, oranges, and greens, starting with lead chromate as the basic product. Lead chromate ($PbCrO_4$) is a yellow pigment with good color retention, produced by solubilizing litharge (PbO) by reaction with nitric acid then precipitating the lead chromate by the addition of sodium dichromate.The precipitated pigment is carefully washed and dried at a temperature below 90 C. It may or may not be diluted with a co-produced or admixed lead sulfate, or lead silicosulfate.

Basic lead chromate ($PbO \cdot PbCrO_4$) is an orange pigment whose color depends upon the proportions of lead chromate and basic lead chromate that are present. The processing is essentially the same as for the preparation of normal lead chromate, mixing a soluble lead salt such as lead nitrate or lead acetate with soluble sodium chromate or dichromate. The precipitate is then washed and dried. In order to increase the basic lead chromate proportion of the mixture, it is common practice to use more sodium chromate than sodium dichromate.

Molybdenum (molybdate) oranges may be made by coprecipitating lead chromate and lead molybdate, and may contain lead sulfate as well. Chrome green may be made by blending chrome yellows with iron blues.

Chrome yellow is principally used in paints, and its increase in recent years is largely attributable to its application in marking highway traffic lanes. Because of their vivid, bright colors, lead chromate pigments also find use in printing inks.A simplified schematic flow sheet for the manufacture of lead chromate-based pigments is shown in Figure 4.14.

4.3.3.2 Production and Industry Structure--

The traditional uses for lead oxide and pigments are either essentially stagnant or decreasing.

Areas where lead oxide is a starting material for the preparation of other products, e.g., heat stabiizers for plastics, provide the only growth factors. Illustrative are the data on white lead, red lead, and litharge (Table 4.19). White lead has consistently decreased, although it may plateau at about its current levels. Red lead paints appear to have stabilized at the 4,000 to 6,000 metric ton/yr level.

The utilization of litharge (PbO) in ceramics (primarily for glazes) has shown growth in recent years, as has its use in rubber compounding, however its use in oil refining has essentially disappeared. Most of the litharge in the "other" category went to the manufacture of batteries, which was adversely affected by the economic slowdown in 1975.

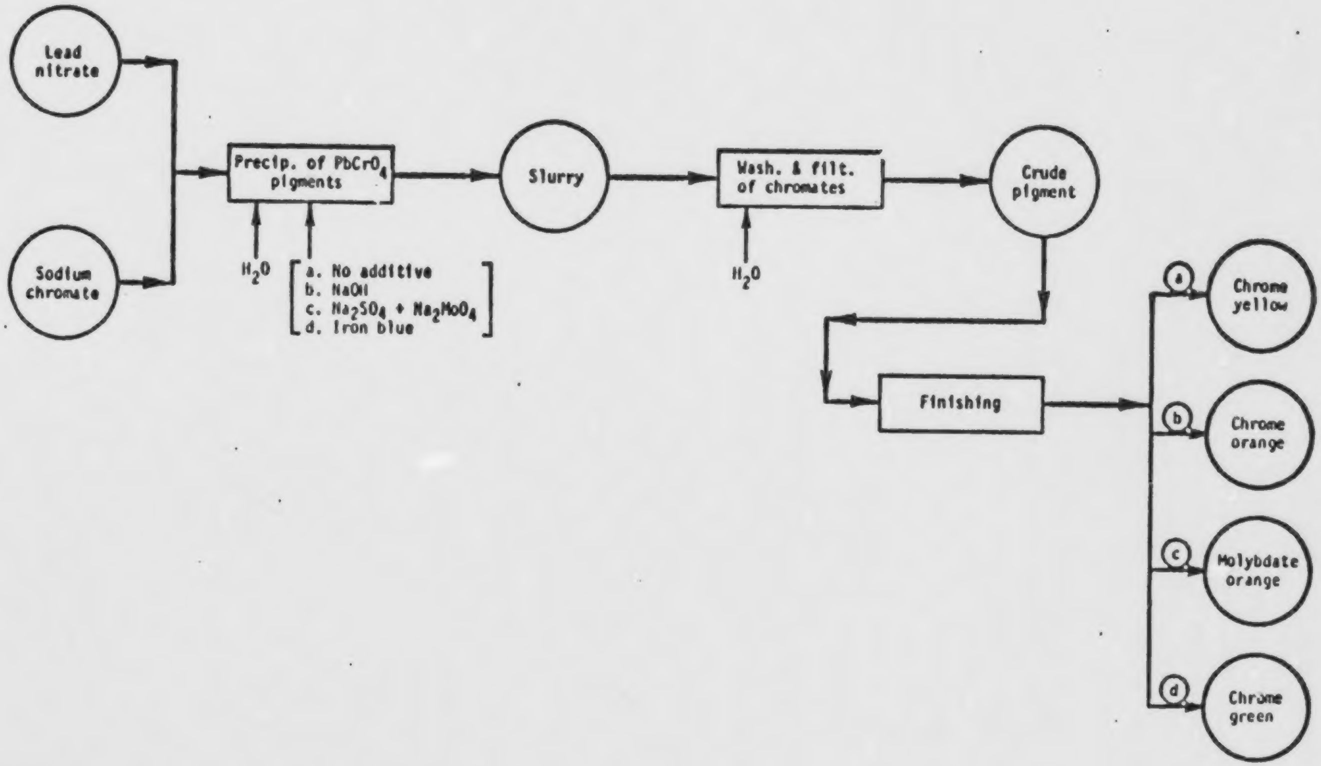


Figure 4.14. Simplified fow sheet of processes for manufacture of lead chromate-based pigments.

Source: Battelle-Columbus (1975).

TABLE 4.19. LEAD PIGMENTS - U.S. SHIPMENTS, BY INDUSTRY, 1971-1975[a]

	Thousands of Metric Tons				
	1971	1972	1973	1974	1975
White lead (dry and in oil)	3,987	6,138	2,900	--	--
Paints	3,987	6,138	2,900	--	--
Ceramics	31	28	16	--	--
Other	2,132	2,963	5,739	5,355	3,021
Total	6,150	9,129	8,655	5,355	3,021
Red lead					
Paints	7,906	4,452	5,903	4,847	4,129
Storage batteries	W[b]	W	W	W	W
Other	11,130	13,482	6,629	7,207	9,562
Total	19,036	17,933	12,532	12,054	13,691
Litharge					
Ceramics	22,074	21,031	32,570	42,264	30,784
Insecticides	W	W	W	W	W
Oil refining	1,282	1,145	562	694	W
Paints	2,798	6,636	2,823	4,850	2,946
Rubber	1,887	1,961	4,606	5,886	5,306
Other	106,054	103,120	121,923	92,736	70,234
Total	134,095	133,893	162,383	146,430	109,270

[a] Source: US Bureau of Mines (1976).

[b] W = Withheld to avoid disclosing individual company confidential data; included with "other".

The major producers and plants are listed in Table 4.20; the number of companies producing these pigments is down to less than a dozen.

4.3.3.3 Future Projections--

The U.S. Bureau of Mines forecast for lead demand for paints by the year 2000 (Ryan and Hague, 1976) projects a demand ranging between 120,000 and 180,000 metric tons, with 135,000 tons as the most probable demand. (This forecast was made before the legislation establishing a maximum of 0.06 percent lead in consumer product paints, and may therefore now be overly optimistic). The pigments category has shown essentially no growth over the past decade, and this appears likely to continue.

4.3.4 Inorganic Lead Compounds

For the purposes of this report inorganic lead compounds are defined as lead compounds other than oxides or pigments which contain no carbon or no direct attachment of a carbon atom to a lead atom; as a consequence of this definition, several lead compounds that contain organic structures are classified herein as inorganic lead compounds. This definition is consistent with industry usage of the term and will facilitate further discussion.

None of these are individually tracked in Bureau of Mines tabulations, and production and consumption data are uncertain. Almost all of the inorganic lead compounds appear to fall under the "litharge" category, since this is the basic starting material. Except for stabilizers for plastics and dryers for paints most of them are relatively small volume, specialty products.

A number of these compounds and industries in which they are used are listed in Table 4.21. One of the salient features of the chart is that, although there are numerous chemical compounds of lead made and used in the "compounding" industries, the generalized classes of compounded products may be considered as few. Further, it may be seen that some compounded products contain more than one lead compound. For example, paint may contain one lead compound as a pigment and another compound as a drier. Similarly, polyvinyl chloride may be stabilized with one lead compound and colored by another. It may also be noted that the classes of products are not mutually exclusive in that a lead compound used as a pigment may be later used for one of the other uses, i.e., paint, dyes, inks, textiles, and/or printing.

With the exception of uses in batteries, the uses shown in the chart are all dissipative, i.e., the lead in these compounds is never recycled, but is diffused through the environment by processes of weathering, wear and erosion, or is disposed of as a component of discarded materials.

TABLE 4.20. PLANTS PRODUCING LEAD PIGMENTS[a]

White Lead	
Lead Carbonate	
Dimensional Pigments, Inc.	Bayonne, New Jersey
NL Industries, Inc. Industrial Chemicals Div.	Chicago, Illinois Oakland, California
Richardson-Merrell, Inc J.T. Baker Chemical Co. (Subsidiary)	Phillipsburg, New Jersey
Smith Chemical & Color Co. Inc.	Jamaica, New York
Whittaker Corp. Rona Pearl Div.	Bayonne, New Jersey
Lead Sulfate, tribasic	
NL Industries Inc. Industrial Chemicals Div.	Perth Amboy, New Jersey Philadelphia, Pennsylvania St. Louis, Missouri Oakland, California
Lead Silicates	
Eagle-Picher Industries, Inc. Chemicals & Fibers Div.	Joplin, Missouri
NL Industries, Inc. Industrial Chemicals Div.	Perth Amboy, New Jersey Philadelphia, Pennsylvania St. Louis, Missouri
Colored Pigments	
Red Lead (Orange mineral)	
Eagle-Picher Industries, Inc. Chemicals & Fibers Division	Joplin, Missouri
Mallinckrodt Chemcial Works Industrial Chemicals Division	St. Louis, Missouri
NL Industries, Inc. Industrial Chemicals Div.	Atlanta, Georgia Charleston, West Viriginia Chicago, Illinois Dallas, Texas Oakland, California Philadelphia, Pennsylvania St. Louis, Missouri Los Angeles, California

TABLE 4.20. (Continued)

Colored Pigments	
Red Lead	
RSR Corporation Quemetco, Inc., subsidiary	Indianapolis, Indiana Middletown, New York Seattle, Washington City of Industry, California
Lead oxide, yellow (litharge)	
American Smelting and Refining Company	Denver, Colorado
Eagle-Picher Industries, Inc. Chemicals and Fibers Division	Joplin, Missiouri
NL Industries, Inc Industrial Chemicals Div.	Atlanta, Georgia Charleston, West Virginia Chicago, Illinois Dallas, Texas Oakland, California Philadelphia, Pennsylvania St. Louis, Missouri Los Angeles, California Portland, Oregon
RSR Corporation Quemetco, Inc., subsidiary	Indianapolis, Indiana Middletown, New York Seattle, Washington City of Industry, California
Lead Chromate	
Hercules Incorporated Coatings & Specialty	Glens Falls, New York
Kewanee Oil Company Harshaw Chemical Company, division Pigment and Ceramic Department	Louisville, Kentucky
Mineral Pigments Corporation Chemical Color Division	Beltsville, Maryland
Richardson-Merrell, Inc. J.T. Baker Chemcial Co. (subsidiary)	Phillipsburg, New Jersey

---

[a] Source: Directory of Chemical Producers, Stanford Research Institute (1976)

TABLE 4.21. INORGANIC LEAD COMPOUNDS AND THEIR USES

Compound	Pigments	Paints	Dyes	Textiles	Printing	Glass	Ceramics	Explosives, Matches	Batteries	Rubber	Plastics	Electronics	Electroplating
Acetate		X	X	X									
Antimonate	X					X	X						
Azide								X					
Beta Resorcylate		X											
Borate		X				X	X						
Carbonate		X					X						
Chloride	X		X										
Chlorosilicate											X		
Chromate	X	X	X	X			X						
Cyanamid		X											
Fluoborate													X
Fumarate										X	X		
Hydroxy Neodecanoate		X									X		
Iodide	X				X								
Isocarboxylate		X											
Linoleate		X											
Maleate											X		
Manganese naphthenate		X											
Manganese tallate		X											
Molybdate	X	X											
Naphthenate		X											
Neo-deconoate		X									X		
Nitrate								X					
Oleate		X											
Orthophosphate	X										X		
Oxides	X	X	X				X	X	X	X	X		

TABLE 4.21. INORGANIC LEAD COMPOUNDS AND THEIR USES (Continued)

Compound	Pigments	Paints	Dyes	Textiles	Printing	Glass	Ceramics	Explosives, Matches	Batteries	Rubber	Plastics	Electronics	Electroplating
Phosphate		X									X		
Phosphate, dibasic											X		
Phthalate, dibasic											X		
Salicylate											X		
Selenide												X	
Silicate						X	X						
Silico sulfate											X		
Silico chromate											X		
Stannate							X						
Stearate		X									X		
Stearate, dibasic		X											
Styphnate								X					
Sulfate	X	X		X	X				X				
Sulfide												X	
Tallate		X											
Telluride												X	
Thiocyanate			X	X				X					
Titanate	X	X					X						
Tungstate	X												
2-Ethylhexanoate		X									X		
Vanadate	X												
Zirconate	X												

4.3.4.1 Manufacturing--

In general, the inorganic lead compounds are produced by typical, non-proprietary processes as follows:

(a) Litharge is reacted with an acid or soluble salt of an acid to produce the desired product. Products that can be produced in this manner include the following lead compounds: acetate, benzoate, borate, cyanide, formate, nitrate, perchlorate, subacetate, and tribasic lead sulfate.

(b) A soluble lead salt (often lead acetate) is reacted with an acid or a soluble salt of an acid often to form a precipitate which is then filtered, washed, and dried. The soluble lead salt may be formed in situ, such as by adding a small quantity of acetic acid to litharge. Products that can be produced in this manner include the following lead compounds: arsenate, benzoate,borate, bromide, carbonate, chloride, citrate, fumarate, iodate, iodide, lactate, maleate, metaborate, molybdate, oxalate, phosphate, dibasic phosphate, phosphite, phthalate, pyrophosphate, salicylate, sesquichloride, stannate, succinate, sulfate, tartrate, thiocyanate, thiosulfate, and tungstate.

(c) Litharge may be fused with an oxide of the desired metallic compound. Products that can be produced in this manner include the following lead compounds: bisilicate, chlorosilicate (by post treatment with hydrochloric acid), metavanadate,molybdate, silicate, silico sulfate, silico chromate (by post treatment with sulfuric or chromic acid, respectively), stannate,titanate, zirconate, and zirconate titanate.

(d) Paint dryers may be manufactured by a fusion process wherein litharge or a soluble lead salt is added slowly to the acid form of the organic material, such as a soap. The temperature is allowed to rise as the lead compound is added; the reaction mass is then externally heated to 110 to 150 C to complete the reaction and to evaporate water from the product. The product may then be filtered, cooled, and packaged, or alternatively filtered, cooled, blended with solvent, and then packaged.

(e) Paint dryers may also be prepared by a precipitation process, by the addition of a soluble lead salt (i.e., lead acetate or nitrate) in water to the soluble sodium soap of a fatty acid in water to form a "precipitate" of an insoluble lead dryer. (In the case of lead naphthenate, the "precipitate" coagulates and floats.) The water layer is removed (decanted) and the product is washed with water and filtered. Finally, the lead dryer is heated to 110 to 150 C to dry the product, cooled, and packaged. It may also be blended with solvent.

(f) Paint dryers may also be produced by dissolving finely divided metal in heated organic acids (100 to 200 C) over 4 to 12 hours using catalysts, such as air, air and water, or acetic acid. Volatilized material is refluxed during the reaction. After the reaction is complete, the catalyst is allowed to evaporate and the product is filtered, cooled, and packaged or blended with solvent and packaged.

(g) Other specific processes:

  (1) Lead iodide may be produced by the reaction of lead metal with iodine.

  (2) Lead bromide may be produced by reacting bromide with lead iodide.

  (3) Lead nitrate may be prepared by the reaction of nitric acid with metallic lead or with several of the oxide forms of lead.

  (4) Lead tetraacetate may be formed by the reaction of red lead with glacial acetic acid.

  (5) Lead fluoride or tetrafluoride may be produced by the reaction of lead carbonate with hydrogen fluoride.

Descriptions of processes employed to produce lead acetylacetonate, fluoborate, hydroxide, and mononitroresorcinol were not identified.

4.3.4.2 Production and Industry Structure--

Although there is some overlap, the inorganic lead chemicals industry can be subdivided into three general groups: lead stabilizers, paint driers, and miscellaneous lead chemicals (frequently small-volume specialty products).

4.3.4.2.1 Lead stabilizers--In the manufacture of some plastics, particularly polyvinyl chloride (PVC) types, it is necessary to heat them to relatively high temperatures to soften them during fabrication operations. The amount of heat that can be applied is limited in degree and duration by the tendency of the compounds to decompose, resulting in some deterioration in physical properties, accompanied by a progressive darkening in color. Additive compounds which promote heat stabilization include metal soaps, organotins, lead compounds, nitrogen compounds, organophosphites, epoxies, and phenols (Weinberg, 1976).

Lead has been employed as a stabilizer for plastics for a longer period than any other product. Initially, lead carbonate was used. Later tribasic lead sulfate, lead silicate, dibasic lead stearate, dibasic lead phthalate, lead stearate, dibasic lead phosphite, and tribasic lead maleate were introduced.

Lead stabilizers have good heat and weather resistance. They are low in price, their good workability ensures high productivity, and they exhibit good dielectric properties (low volume resistivity) and low water absorption. They are used extensively in PVC electrical wire and cable insulation and in opaque rigid PVC extruded and injection molded products, where they confer weather resistance as well as protection against heat during molding. Sulfur staining, lack of clarity, and toxicity of lead stabilizers are some of their drawbacks. (Weinberg, 1976).

Typically, plastics formulations may contain about 0.5 percent lead. At one time a few producers used lead stabilizers in DWV (drain, waste, and vent) pipe, but since Schedule 40 plastic pipe may also be used for potable water piping, this application has been abandoned. Industry sources state that no lead is used in any PVC pipe. If any plasticizer is used it is more likely to be 0.1 to 0.2 percent of organotin (Furno,1978).

The use of lead stabilizer in PVC pipe for potable water service is effectively precluded by National Sanitation Foundation Standard No. 14 (1965). This standard, applicable to thermoplastic pipe, fittings, valves, traps, and joining materials, specifies a maximum lead concentration of 0.05 mg/l after a 72-hour leach in 1 percent nitric acid, which will be exceeded if a lead stabilizer is present (National Sanitation Foundation, 1977).

A list of producers of lead-based stabilizer materials utilized in the manufacture of plastics and rubber products is presented in Table 4.22. The major manufacturer of the lead based plastic and rubber types of stabilizer materials presented in Table 4.22 is NL Industries, which dominates sales of these materials, accounting for about 67 percent of the business. Other significant suppliers include Eagle-Picher, Gulf Resources (Bunker Hill Division), American Cyanamid (MacGregor Lead Division) and Hammond Lead Products (Halstab Division). Although F. D. Davis has a significant raw material position, it is believed that this company sells most of its material for pigment use. It is not believed that Dimensional Pigments, Rona Pearl, or Smith Chemical sell significant quantities of materials for stabilizer use. Finally, J. T. Baker and City Chemical probably only sell reagent grade material for other uses.

Consumption of the lead compounds shown in Table 4.22 in the plastics and rubber industries in 1974 has been estimated as presented in Table 4.23. Their use in plastics has been in a gradual decline in recent years, perhaps prompted by increasing environmental regulations, as evidenced by one industry estimate of 1973-1976 consumption:

TABLE 4.22. PRODUCERS OF LEAD-BASED PLASTIC AND RUBBER STABILIZER-TYPE MATERIALS[a]

Company[b]	Location	Lead Stabilizer												
		Carbonate	Chlorosilicate	Fumarate, tetrabasic	Maleate	Phosphate	Phosphate, dibasic	Phthalate, dibasic	Salicylate	Silicates	Silico sulfate	Silico chromate	Sulfate	Sulfate, tribasic
American Cyanamid & Company	Wayne, NJ	X												X
MacGregor Lead Division	Chicago, IL	X												
City Chemical Company[c]	Jersey City, NJ	X											X	
F. D. Davis Company[c]	South Plainfield, NJ	X												
Dimensional Pigments[c,d]	Bayonne, NJ	X												
Eagle-Picher Industries	Joplin, MO									X		X		
	New York, NY									X				
	Cincinnati, OH	X												X
Gulf Resources & Chemical Corp.	South Plainfield, NJ	X												
Bunker Hill Division														
Hammond Lead Products Company	Hammond, IN	X											X	X
Halstab Division														
NL Industries[e]														
Industrial Chemicals Div.	Charleston, WV									X				
	Philadelphia, PA		X	X	X		X	X	X		X			X
	St. Louis, MO										X	X		
Metals Division	Chicago, IL	X												
Richardson-Merrell, Inc.[c]	Phillipsburg, NJ	X											X	
J. T. Baker Chemical Co.														
Rona Pearl, Inc.[d]	See footnote e					X								
Smith Chemical & Color Co.[d]	Jamaica, NJ	X								X				

[a] Source: Chemical Economics Handbook, Stanford Research Institute (1975).

[b] Active stabilizer suppliers to the trade include Argus (Witco subsidiary), American Cyanamid, Eagle-Picher, Ferro, Halstab, Interstab, Mooney, NL, Tenneco, Witco, RT Vanderbilt Company, Inc., American-Hoescht, and M.R.S. Chemicals. It is believed that Vanderbilt, Hoechst, and M.R.S. do not manufacture such compounds in the United States.

[c] Believed to be a producer of pigment or reagent grade only and not sold as stabilizer.

[d] Carbonate to supply Dimensional Pigments and Rona Pearl is produced at one plant.

[e] The Oakland, California plant was shut down prior to 1977 and the Perth Amboy plant was shut down March 15, 1977.

TABLE 4.23. ESTIMATED CONSUMPTION OF LEAD-BASED STABILIZERS IN PLASTICS AND RUBBER INDUSTRIES, 1974[a]

	Millions of kg (lb)
Tribasic lead sulfate	7.3 (16)
Chloro silicates Dibasic lead stearate[b] Blends of chlorosilicates and dibasic lead stearates[b]	3.6 (8)
Lead carbonate	2.7 (6)
Dibasic lead phthalate	2.3 (5)
Dibasic lead phosphate	1.4 (3)
Silico-chromate	0.5 (1)
Silico sulfate	0.5 (1)
Fumarate, maleate, phosphate, salicylate, silicate, and bisilicate	Negligible
Total	18.3 (40)

[a] Source: Battelle - Columbus estimate, based on industry estimates.

[b] Actual consumption of dibasic lead stearate is small.

Consumption of Heat Stabilizers
(In Metric Tons)

Stabilizer	1973	1974	1975	1976
Barium-Cadmium	23,000	22,300	17,500	22,000
Lead	14,600	14,600	6,400	8,200
Calcium-zinc	1,360	1,400	1,000	1,300
Tin	7,600	7,500	5,800	7,000
Total	46,560	41,600	30,700	38,500

Source: Modern Plastics (1976).

4.3.4.2.2 Paint driers--Producers of paint driers are listed in Table 4.24. Major producers of lead-based driers presented in Table 4.24 include Ferro,Interstab, Mooney, Norac,Shepherd, Sherwin-Williams, Tenneco,Troy, and Witco; the relative positions of Diamond Shamrock, NL Industries, and Smith Color are believed to be small. The major manufacturers of these driers produce other metallic driers in the same or similar equipment and, thus, capacity figures are not meaningful.

Historical production rates of selected lead driers are presented in Table 4.25. Total production of the three most popular lead driers peaked in 1969 at more than 10 million kg (23 million pounds). Drier production then entered a period of decline, as water-based latex paints more and more supplanted traditional oil-base paints. Additionally, more recently the restrictions on the use of lead in paints have caused the replacement of lead driers by other materials. By 1975, total production of lead-based driers had declined to about 3 million kg (6.5 million pounds) or to 28 percent of the peak production rate. Lead consumed in these products in 1975 was about 1,360 metric tons (1,500 short tons), when total sales revenue of such driers were estimated to have been about 4.5 to 5.0 million dollars.

As is noted in the footnotes to Table 4.24 some of the lead 2-ethyl hexanoate, hydroxy neodecanoate, neodecanoate, stearate, and dibasic stearate produced by Ferro, Interstab, Mooney, NL Industries, Tenecco, and Witco are also used as rubber and plastic stabilizers.

4.3.4.2.3 Miscellaneous lead compounds--A number of lead compounds are produced in limited quantity or to industrial order for special applications. Some of these are produced by more than one manufacturer (Table 4.26); others by only one manufacturer (Table 4.27). The diversity of the inorganic lead compound industry is illustrated by the fact that in the tables contained in this section 46 companies are shown, producing 60+ chemicals at 50+ locations.

TABLE 4.24. PRODUCERS OF LEAD-BASED PAINT DRIERS

Company	Location	Lead Drier Compound													
		2-Ethyl hexanoate b	Isocarboxylate	hydroxy neodecanoate b	Linoleate	Manganese neodecanoate b	Manganese tallate	Naphthanate	Neodecanoate b	Oleate	Oleate-linoleate	Beta resorcylate	Stearate b	Dibasic stearate b	Tallate
Diamond Shamrock Corp. Process Chemicals Division	Cedartown, GA												X	X	
Ferro Corp. Ferro Chemical Div.	Bedford, OH	X						X							X
Interstab Chemicals, Inc.	New Brunswick, NJ	X				X		X			•				X
Mooney Chemicals, Inc.	Franklin, PA	X	X	X			X	X	X						X
NL Industries, Inc. Industrial Chemicals Division	Philadelphia, PA													X	
Notac Company, Inc. Mathe Division	Lodi, NJ							X		X			X	X	
Shepherd Chemical Co.	Cincinnati, OH	X			X			X	X	X		X			X
Sherwin-Williams Co.	Cleveland, OH							X							
	Emeryville, CA							X							
	Garland, TX							X							
Smith Chemical and Color Company	Jamaica, NY												X		
Tenneco Inc.	Elizabeth, NJ	X						X		X					X
Tenneco Chemicals	Long Beach, CA	X						X		X					X
Troy Chemical Corp.	Newark, NJ	X			X			X			X				X
Witco Chemical Corp.	Clearing, IL	X						X					X		
Organics Division	Lynwood, CA	X						X							

[a] Source: Chemical Economics Handbook, Stanford Research Institute (1975).

[b] Several lead compounds classified herin as paint driers are used as lead stabilizers including 2-ethyl hexanote, hydroxy neodecanoate, nedecanoate, stearate, and dibasic stearate.

TABLE 4.25. PRODUCTION OF SELECTED LEAD-BASED PAINT DRIERS[a]

	Production, millions of kg (lb)			
Year	Naphthenate	2-Ethyl Hexanoate	Tallate	Total
1966	6.47 (14.27)	0.11 (0.24)	1.64 (3.61)	8.22 (18.12)
1967	6.06 (13.37)	0.09 (0.19)	1.62 (3.58)	6.41 (14.14)
1968	7.73 (17.04)	0.47 (0.82)	1.78 (3.92)	9.88 (21.78)
1969	8.19 (18.06)	0.39 (0.87)	1.99 (4.38)	10.57 (23.31)
1970	6.51 (14.36)	0.69 (1.52)	1.90 (4.16)	9.09 (20.04)
1971	5.48 (12.08)	0.20 (0.45)	1.18 (2.60)	6.86 (15.13)
1972	5.19 (11.45)	1.57 (3.47)	0.91 (2.00)	7.67 (16.92)
1973	5.26 (11.61)	0.40 (0.89)	0.60 (1.33)	6.27 (13.83)
1974	4.10 (9.04)	1.11 (2.44)	0.51 (1.13)	5.72 (12.61)
1975	1.87 (4.12)	0.80 (1.77)	0.42 (0.65)	2.97 (6.54)

[a] Sources: Current Industrial Reports, Series M28A, U.S. Department of Commerce, Bureau of the Census 1966 through 1973.

Synthetic Organic Chemicals, U.S. Production and Sales, U.S. International Trade Commission.

TABLE 4.26. PRODUCERS OF MISCELLANEOUS LEAD-BASED CHEMICALS PRODUCED BY MORE THAN ONE COMPANY[a]

Company	Location	Lead Product							
		Arsenate [b]	Fluoborate	Iodate	Iodide	Nitrate	Subacetate	Thiocyanate	Zirconate Titanate
Allied Chemical Corporation	Marcus Hook, PA						X		
C. P. Chemicals, Inc.	Swaren, NJ		X						
Chempar Chemical Company	Portland, OR[c]	0							
City Chemical Corporation	Jersey City, NJ				X			X	
Deepwater Chemical Company	Irvine, CA			X	X				
Dimensional Pigments, Inc.	Bayonne, NJ[d]	0							
Harstan Chemical Corporation	Brooklyn, NY		X						
Hummel Chemical Company	South Plainfield, NJ							X	
Kewanee Industries	Cleveland, OH		X[e]						
Harshaw Chemical Company									
Los Angeles Chemical Company		0							
Mallinckrodt, Inc.	St. Louis, MO					X			
NL Industries	Niagara Falls, NY								X
Industrial Chemicals Division									
R.S.A. Corporation	Ardsley, NY			X	X				
Richardson-Merrell, Inc.	Phillipsburg, NJ					X	X		
J. T. Baker Chemical Company									
Rona Pearl, Inc.	Bayonne, NJ[c]	0							
G. Frederick Smith Chemical Company	Columbus, OH					X			
Transelco, Inc.	Pennyan, NY								X
Woolfolk Chemical Works, Inc.	Fort Valley, GA	0							

[a] Source: Chemical Economics Handbook, Stanford Research Institute (1975).

[b] No lead arsenate is being produced at the present time according to these companies.

[c] Until recently, Chempar had acquired its lead arsenate from Woolfolk.

[d] Product is (or was) produced in one plant to serve both Rona Pearl and Dimensional Pigments.

[e] It is believed that Harshaw no longer produces lead fluoborate.

TABLE 4.27. MISCELLANEOUS LEAD-BASED CHEMICALS PRODUCED BY ONLY ONE COMPANY.

- City Chemical Corporation (Jersey City, NJ)
  - benzoate
  - bromide
  - citrate
  - cyanide (metallurgy reagent)
  - hydroxide
  - lactate
  - metavanadate
  - molybdate
  - oxalate
  - pyrophosphate
  - sesquichloride
  - stannate (ceramic electronic bodies)
  - succinate
  - tartrate
  - thiosulfate (rubber accelerator)
  - tungstate (pigment)
- MacKenzie Chemical Works (Central Islip, NY)
  - acetylacetoate
- NL Industries
  - Philadelphia, PA - formate (rubber compounding)
  - Niagara Falls, NY - titanate (pigment and ceramics)
    - zirconate (electronic devices)
- Pennwalt Corporation, Ozark-Mahoning Co. (Tulsa, OK)
  - fluoride
  - tetrafluoride (potential fluorinating agent for hydrocarbons)
- Olin Corporation, Winchester (Niagara Falls, NY and East Alton, IL)
  - styphnate (detonator for explosives)
- Richardson-Merrell, Inc., J. T. Baker Chemical Company (Phillipsburg, NJ)
  - chloride (artist pigment, solder, and solder flux)
- G. Frederick Smith (Columbus, OH)
  - perchlorate (analytical reagent)
- Syntex Corporation, Arapahoe Chemicals Division (Boulder, CO)
  - tetraacetate
- Thiokol Corporation, Ventron Corporation, Alfa Products Division (Danvers, MA)
  - telluride
- Tyler Corporation, Atlas Powder Company (Tamaqua, PA)
  - nitroresorcinol

[a] Source: Directory of Chemical Producers, Stanford Research Institute (1976).

Lead fluoborate, used in electroplating in negligible quantities, is producd by electroplating supply firms including C. P. Chemicals, and Harstan, but probably is no longer produced by Harshaw (Kewanee Industries). Lead iodide is produced in small quantities for photographic use and as a gold or bronze pigment by Deepwater Chemical R.S.A. Corporation. (These companies also produce lead iodate for unknown uses). City Chemical can also produce reagent grade iodide for specialty use.

Although lead nitrate is a water soluble form of lead that would be useful for preparation of other lead materials or for match manufacture, pyrotechnics, and dyeing, only Mallinckrodt, J. T. Baker, (Richardson-Merrell) and G. Frederick Smith reportedly manufacture the product, probably only reagent grade and in very small quantities.

Allied Chemical Corporation and J. T. Baker (Richardson-Merrell) produce lead subacetate in minor quantities for sugar analysis. Similarly, lead thiocyanate is produced in small quantities as a primer for explosives and matches by Hummel Chemical Company and City Chemical.

Lead zirconate-titanate is produced in small quantities by NL Industries and Transelco and is used as a dielectric.

Of the lead products listed in Table 4.27, lead styphnate (produced by Olin for use as a detonator), lead chloride (produced by J. T. Baker for use in artist pigments and solder), and lead tetraacetate (produced by Arapahoe for textile dyeing) are the only materials that might be produced in any significant quantity. Most materials produced by City Chemical are used for reagent or for specialty purposes. Acetylacetonate is produced for an unknown and probably specialty purpose by MacKenzie.

Less than 100 tons per year each of lead formate, lead titanate, and lead zirconate are produced by NL Industries.

Consumption of the lead fluoride compounds produced by Ozark-Mahoning (Pennwalt) is believed to be very small and for specialty purposes. G. Frederick Smith produces lead perchlorate as a reagent chemical. No significant uses for the nitroresorcinol and telluride produced by Syntex and Alfa (Thiokol) are known.

In past years, lead arsenate was a popular insecticide, e.g., it was the insecticide of choice for protecting apple orchards against the coddling moth. The quantities produced and used were significant, as evidenced by the following U.S. Department of Agriculture (1975) tabulation:

Year	Metric Tons
1967	2,700
1968	4,090
1969	4,170
1970	1,880
1971	2,800
1972	2,340
1973	1,790
1974	W *
1975	--

---

w withheld to avoid disclosure of individual companies.

* now combined with all inorganic insecticides, fungicides, and rodenticides

Since World War II, lead arsenate has been supplanted by various organic insecticides. This trend was accentuated by concern of the FDA for lead in foods, particularly in canned apple juice and applesauce for infants. The refusal of food packers to accept apples sprayed with lead arsenate effectively terminated its use in the U.S. A survey of all the companies listed in Table 4.23 as lead arsenate producers revealed that all manufacturers claim not to be a current producer. Also, no record of importation of lead arsenate was located. Most recent status of lead arsenate, as determined by EPA (U.S. Environmental Protection Agency.1978) is that none is being manufactured in the United States, and that sales, if any, are less than 500 kg/yr of active ingredient.

This use was obviously a totally dispersive one, and considerable residues of lead arsenate have been shown to have accumulated in orchard soils (See Section 5.2.3).

4.3.4.3 Future Projections--

It is evident from the preceding discussion that the production of inorganic lead compounds is a minor segment of the lead industry whose future will be influenced primarily by the effects of environmental considerations rather than by the growth prospects of the products in which they are used, or their effectiveness in those uses. Thus, the projection for these compounds is for stagnant or declining markets.

4.3.5 Miscellaneous Uses of Metallic Lead

Lead has a variety of uses in metallic form, either singly, or alloyed with other metals. Most of these uses derive from one or more of the special attributes of metallic lead--high density, corrosion resistance, malleability, castability, and low melting point.

The components of the metal end-use category, as compiled annually by the Bureau of Mines (See Table 4.11), consist of a heterogenous collection of products, alloys, and mill shapes. Several are specific--i.e., ammunition, bearing metals, cable covering, type metal, collapsible tubes, and the construction-oriented pipes, traps, bends, and caulking lead. The remainder designate alloys or mill product forms, each of which has a variety of applications--viz., brass and bronze, casting metals, foil, sheet lead, solder, and terne metal.

Uses of metallic lead have been declining in the post-World War II period. From a level of 360,000 metric tons in 1966, these gradually fell to about 300,000 tons in 1974, a loss of 60,000 tons in 8 years, and then plummeted a further 60,000 tons in one year to about 240,000 tons in 1975, continuing at this level in 1976 (Table 4.10).

These end uses, except for ammunition, are potentially non-dissipative, although reclamation and recycle of the product items are erratic; the larger the physical size of the item, the higher the probability of recycle. Also, a closed loop with recycle is a normal part of some uses, such as for cable covering and type metal, and added lead is primarily for makeup.

These miscellaneous uses are described briefly in the following pages, in generally descending order of annual consumption.

4.3.5.1 Ammunition--

Lead continues to be the major metal used for sporting ammunition in the form of shot and small-caliber rifle and handgun bullets. The alloy used may contain up to one percent arsenic for shot, and up to 2 percent antimony for bullet cores. Lead ammunition is still used in military training (Ryan and Hague, 1976).

Except for target range shooting, where reclamation is feasible and frequently practiced, ammunition would have to be classed as a dissipative use. A particular problem with lead shotgun shot is that deposited on the bottoms of ponds and marshes, where it is available to waterfowl. It is being replaced for waterfowl hunting by steel shot, as discussed later.

Use of lead for ammunition declined from 79,000 metric tons in 1974 to under 67,000 metric tons in 1976. Future projection of the U.S. Bureau of Mines (Ryan and Hague, 1976) gave a forecast base of 100,000 short tons (91,000 metric tons) in the year 2000, on a basis of paralleling population growth. In view of the declining trend of the last several years, and the proposed restrictions on the use of lead shot for waterfowl hunting, this estimate is considered to more probably represent the upper range of the forecast. Lead consumption over the near term is believed to more likely fall in the 70,000-80,000 metric ton range.

4.3.5.2 Solder--

One of the commonest lead alloys is solder, the designation for a series of relatively low-melting alloys of lead and tin. The half-and-half grade (50 percent lead and 50 percent tin) is a general all-purpose grade. Principal uses of solder are to form water-tight or electrically conductive joints or to fill surface imperfections on automobile or appliance surfaces. Food can manufacture, electronic component assembly, and automobile manufacture are the major end-use categories. The quantity of solder used per automobile is considerable; the average has been estimated to be of the order of 10 kg. Solder is used in radiator fabrication and for smoothing body surfaces at joints in manufacture. Its use in body repair work is declining as competition from epoxy cold-set filling compounds intensifies.

Common solder alloys range from the low melting (183-192 C) 70 percent tin - 30 percent lead alloy used for coating metals, to 2 to 5 percent tin, 95 to 98 percent lead alloys, used for soldering cans. A 60-40 grade is used for general purposes, but particularly where the temperature requirements are critical. The 50-50 grade, a general purpose grade, is most popular of all. The 40-60 alloy is a plumbers wiping solder for joining lead pipes and cable sheaths, and the 20-80 solder is used for filling dents or seams in automobile bodies (American Society for Testing Materials, 1978b).

Lead consumption for solder has fallen off irregularly from the 71,560 metric tons used in 1966, as indicated by U.S. Bureau of Mines data tabulated below:

1966	71,560	1972	64,620
1967	63,340	1973	65,095
1968	67,180	1974	60,015
1969	65,870	1975	52,010
1970	63,220	1976	57,430
1971	63,500		

Automated machinery has permitted a reduction in the amount of solder needed for a given joint and alternative technologies--e.g., adhesive or welded can seams, aluminum cans, crimp and clamp electrical connections, epoxy body seam fillers, etc. have contributed to reductions in lead usage.

The forecast is for this contraction in the consumption of solder to continue, fueled by the increasing concern about the health effects of lead and the tightening regulatory climate. One particularly vulnerable segment is the solder used in joining seams in food cans, estimated to have consumed approximately 4,000 metric tons of lead in 1976 (U.S. Bureau of Mines, 1978c). The environmental effects of the use of soldered food cans are discussed at length in Chapter 8.

4.3.5.3 Sheet Lead--

Sheet lead is used to line chemical equipment for corrosion resistance, to fabricate radiation shielding in X-ray rooms and wherever radioactive materials are used or handled, and as a sound barrier or absorber. Sheet lead is used also in building construction as a roofing material, for flashing for roof vents, and for shower stall pans.

Consumption of sheet lead is slightly lower than it was a decade ago, but may be in the process of reversing that downtrend. Contrary to most lead uses, 1975 consumption increased over that of 1974, and 1976 consumption held that gain (Table 4.11). The increasing emphasis by OSHA on sound levels in industrial situations is expected to accentuate the use of sheet lead for sound absorption.

The use of metallic lead in sheet form is not a particularly dissipative use. The weight and value of sheet lead used in a given industrial application is such as to favor recovery and recycle when the equipment or building is retired and scrapped.

4.3.5.4 Cable Covering--

Cable coverings use lead in either of two distinct ways. Lead alloys are extruded around assembled cables for underground or underwater installation. Connections between cable sections are built up in place (underground) or on the deck of a cable-laying vessel from thin-gage sheet lead and sealed to the extruded covering of the cable lengths. Intercontinental cables continue to rely on lead but plastic coverings have supplanted lead in recent years in less critical services. Cable sheathing is reclaimable as old scrap when the cable is replaced--5 to 25 years after installation.

The second usage of lead in cable production is as a sheath for retention of rubber-based coatings during curing. In this application, soft unalloyed lead is extruded onto the uncured rubber covering of the assembled cable and retained during a steam curing cycle, then stripped and recycled to the melting tank for reuse. This is basically a non-dissipative use, requiring additional lead only to make up for handling losses, e.g., drosses resulting from oxidation, which are removed from the melting pot and recycled as lead scrap.

The decrease in the consumption of lead for lead-sheathed cable since 1966 has been dramatic, decreasing from over 60,000 metric tons to less than 15,000 in 1976 (U. S. Bureau of Mines data):

1966	60,310	1972	41,660
1967	57,170	1973	39,000
1968	48,480	1974	39,390
1969	49,160	1975	20,045
1970	46,050	1976	14,450
1971	48,000		

This decrease is believed to reflect the increasing substitution of other (plastic) coverings for lead. The greater reliance on microwave domestic transmission and transmission via satellite for overseas transmission in place of underwater cables is a contributing factor; this trend is likely to continue. No expansion in this use is forecast.

4.3.5.5 Weights and Ballast--

Weights and ballast is a general subcategory consuming substantial amounts of lead annually. Sailing vessel keels, submarine ballast, and industrial weights for balancing probably are the largest items dimensionally, but automobile tire balancing weights--from one-half ounce to 6 ounces each--account for the most lead. A major portion of the discarded tire weights do not get recycled, but wind up in municipal dumps or land fills. The total weights and ballast subcategory ranged from 14,000 to 19,000 metric tons of lead per year in the 1966 to 1975 decade, trending upward slightly.

4.3.5.6 Type Metal--

Type metal is a lead-based alloy with antimony and tin used by linotype machines in the preparation of printed materials, such as newspapers and books. The line of type is assembled from dies containing the appropriate letter cavities. The dies are locked in place and molten type metal is injected and cooled, forming a slug on the top of which the raised printing characters are located. The slugs are assembled into frames and inserted into a press where the printing takes place. When the printing run is complete, the slugs are returned to the linotype machine and fed into the melter for recycle. These electrically-or gas-heated pots hold up to several tons of molten metal. Surplus ink on the slugs create fumes at the melting pot which are objectionable to the type-setters. Due to the heat and the irritating smoke, the pots are hooded and the area well ventilated.

Recently, new processes have been developed which do not require lead use, the cold type (photo typesetting) and offset presses. Many firms have already changed over to the cold process, and in 5 to 10 years all but 5-7 1/2 percent of the industry will have changed over (Short and Associates, 1976).

Type metal is a non-dissipative use, with total recycle by the user; the only lead consumption is for makeup to replace dross losses and other handling losses.

Consistent with the developments described above, the consumption of lead for type metal has also exhibited a significant decrease since 1966, dropping over 50 percent, from 27,590 metric tons to 13,610 in 1976 (U.S. Bureau of Mines data):

1966	27,590	1972	18,090
1967	25,900	1973	19,880
1968	25,380	1974	18,610
1969	23,270	1975	14,700
1970	22,200	1976	13,610
1971	18,880		

The forecast is for the annual decrease to continue, with consumption possibly leveling off at a few thousand tons per year after 1985.

4.3.5.7 Pipes, Traps, Bends, and Caulking Lead--

Lead pipe, traps, and bends are still specified by some building codes (less for residential construction), and lead pipe is still used for corrosive service by the chemical industry. Lead pipe used industrially is probably reclaimed at the end of its service life; traps and bends in domestic use are reclaimable, but the percentage may not be very high at present. Thus, this use, while not necessarily dissipative, probably results in a solid waste discharge to the environment. On the other hand, lead in this form is quite immobile and resistant to degradation, so that the insult to the environment is minimal.

Alternative materials, e.g., copper, brass, and stainless steel are making major inroads into these traditional uses.

Lead is used to caulk bell joints in cast iron pipe, usually by pouring molten lead into the joint, which has been sealed on the bottom by stuffing oakum down in the joint. Many building codes still require this type of joint, although more modern pipe and joint techniques, e.g., copper pipe and soldered joints, and plastic pipe and cemented joints, are making inroads into lead-caulked cast iron pipe, especially for such applications as vent stacks.

Both of these applications of lead are in declining trends, as shown by the U. S. Bureau of Mines consumption data since 1966:

	Caulking Lead	Pipes, Traps and Bends
1966	57,380	18,120
1967	43,340	18,310
1968	45,100	19,310
1969	40,690	17,600
1970	31,390	16,220
1971	27,200	16,480
1972	20,390	16,130
1973	18,190	19,130
1974	17,900	14,930
1975	12,970	12,910
1976	11,310	12,500

The decline was especially severe, over 80 percent, for caulking lead; that for pipes, traps, and bends was less so, and lead consumption for this purpose may stabilize at about the 12,000 ton/yr level. Where the decline in caulking lead will plateau, if indeed it does, is uncertain. In any case, like so many other segments of the lead industry, there are no prospects for growth for these uses.

4.3.5.8 Brass and Bronze and Bearing Metals--

Brass is basically an alloy of copper and zinc. Lead is an alloying additive used to primarily improve its machinability. "Leaded brass" contains 62.5 percent copper, 35.75 percent zinc, and 1.75 percent lead. In "high lead brass" the lead content is increased to 3 percent; a "low lead brass" would have only 0.5 percent lead. Bronzes are basically alloys of copper and tin; lead is also added to some bronzes. Automobile radiators, plumbing fixtures, building hardware, and corrosion-resistant industrial pipe and fittings are major end uses for these alloys. Competition for brass and bronze in these applications comes principally from plastics, aluminum and stainless steel. Secondary recovery of brasses and bronzes is extensive.

Transportation requires lead in bearings, where its natural qualities of lubrication and resistance to wear are the basis for applications in automotive equipment and railroad car journals (Ryan and Hague, 1976). A common composition for railroad car bearings is 86 percent lead, 5 percent tin, and 9 percent antimony. Copper-lead bearing alloys may contain as much as 40 percent lead. Leaded bronzes are essentially mixtures of lead, copper, and tin, with a lead content of 4 to 25 percent (Metals Handbook, 1961).

Lead bearing metals have been the traditional material for railroad car journal boxes and for line-shaft support bearings. Both applications have had increasing competition from roller bearings and lead consumption continues to decline. Minor wear occurs in such bearings in use but the bulk of the material is recoverable.

The consumption data reported in the Annual Minerals Yearbooks (U.S. Bureau of Mines) illustrate the declining lead demands since 1966 for both of these use categories:

	Bearing Metals	Brass and Bronze
1966	19,580	23,080
1967	17,740	18,560
1968	16,730	19,070
1969	15,790	19,510
1970	14,810	17,170
1971	14,770	18,180
1972	14,340	17,960
1973	14,200	20,620
1974	13,250	20,170
1975	11,050	12,160
1976	11,850	14,200

The consumption of lead for bearing metals has declined steadily and consistently since 1966, and the decline has probably not yet run its course. Brass and bronze fairly steadily demanded 18,000 to 20,000 metric tons of lead per year up to 1974. In 1975 and 1976 lead consumption collapsed to 12,000 to 14,000 tons per year, for reasons not clear. Both uses will be fortunate to hold at present levels, and no growth is forecast for either.

4.3.5.9 Casting Metal--

Casting metals provide easily formed structural parts for nonload bearing industrial machinery and corrosion resistant equipment, such as chemical pumps. Also, the nuclear industry widely utilizes cast lead for lead bricks weighing about 12 kg (27 lb), for lead pigs for the shipment of radioactive materials, and for lining the large casks used to ship spent reactor fuel elements. Most of the lead in this use is recovered and recycled.

Lead consumption for this use category varied irregularly between 6,000 and 9,000 metric tons per year during the past decade, and has ranged between 6,000 to 7,000 tons since 1970. It appears likely to continue at about this level, with essentially no prospects for significant growth.

4.3.5.10 Collapsible Tubes and Foil --

Collapsible tubes of lead pioneered the handy, disposable personal package for toothpaste, creams and ointments, and similar personal care and pharmaceutical products. Since World War II, increasing concern about the health aspects of lead in such tubes has led to the adoption of other materials. Aluminum, along with plastics, with their ready availability, lower price, and better resistance to product discoloration or metal contamination--have become dominant in this field.

Collapsible tubes of lead have become almost obsolete, except for a few specialized uses such as for artists' colors in oil, and other minor industrially-oriented products. After use, the tubes are discarded rather than collected for recycling. Annual lead consumption for these uses has declined about 80 percent from the approximately 11,000 metric tons per year of the late 1960's to a little over 2,000 tons per year since 1973. It gives evidence of stabilizing at about this level, with no prospects for growth.

Lead foil is used to shield radioactive materials (gamma-ray attenuation) during shipment and storage. Lead foil consumption has ranged between 4,500 and 6,500 metric tons per year over the past decade, with no strong trend either up or down; it can probably be expected to continue in similar fashion.

4.3.5.11 Miscellaneous Uses--

There are several miscellaneous uses for metallic lead, each consuming no more than a few hundred to a few thousand metric tons per year. These include annealing, galvanizing, and plating.

As an annealing bath, low-melting lead alloys provide a curing medium for various metals. This is a modest use of lead, 3,700 metric tons in 1974 and 2,400 in 1975, which will probably continue at a nominal level. It appears to have no major growth potential. It is an essentially nondissipative use, with apparently very little impact upon the environment.

Lead has a minor use in plating steel parts, such as nuts and bolts, primarily to provide lubrication and as an anti-galling agent. Terne metal, an alloy of lead and tin, is used to coat steel sheets to provide corrosion resistance. Terne alloy contains 10 to 25 percent tin, with the remainder lead. Lead coatings that contain as little as 2 to 2.5 percent tin are increasingly popular (Metals Handbook, 1961). Major use for terne metal is for automobile gasoline tanks (the inner suface) to prevent rusting of this vital automobile component. Lead consumption for these uses has ranged between 2,000 and 4,000 tons per year during the past decade, and is expected to continue at about this level. No growth is forecast for this segment of the lead industry.

4.3.6 Organic Lead Compounds

For the purposes of this report organic lead compounds are defined as those compounds in which a lead atom is attached directly to a carbon atom. A large number of compounds containing lead and carbon which do not fit this definition were discussed as inorganic compounds in Section 4.3.4.

The major organic lead compounds are, of course, tetraethyl lead and tetramethyl lead; these were discussed in Section 4.3.2.

Although a large number of organic lead compounds theoretically could be produced, and over a thousand are known, only one specific product, tetraphenyl lead, and two manufacturers were identified.

The Houston Chemical Company Division of PPG Industries, produces (or did produce) unspecified organic lead compounds at Beaumont, Texas; the Alfa Products Division of Ventron Corporation (subsidiary of Thiokol Corporation) produces tetraphenyl lead at Danvers, Massachusetts.

The list of possible types of organic lead compounds include:

Hexaorgano dilead	R_6Pb_2
Tetraorgano lead	R_4Pb_2
Triorgano lead halide	R_3PbX
Triorgano lead compound	R_3PbOR'
Di (Triorgano lead) oxide	$(R_3Pb)_2O$
Triorgano lead alkoxide	R_3PbOR_2'
Triorgano lead organophosphide	R_3PbPR_2'
Triorgano lead azide	R_3PbN_3

where R and R' refers to an alkyl, aryl, or alkenyl group, and X refers to an halide. These products may be produced by a variety of processes:

- Reaction of a lead alloy with an organic ester
- Grignard reaction mechanism using lithium or magnesium chloride with an inorganic lead salt
- Reaction of lead salts with trialkyl aluminum
- Lead metal reacted with alkylhalides, phosphates or sulfates
- Lead metal with an alkyl chloride, reacted with magnesium alkyl magnesium halide, alkyl lithium, sodium aluminum alkyl, or sodium boron alkyl
- Electrolysis using a lead anode and a Grignard reagent (analogous to the electrolytic method for producing (TML)
- Trialkyl lead chloride plus lithium aluminum hydride to produce trialkyl lead hydride
- Trialkyl lead hydroxide plus metallic sodium to produce di (trialkyl lead) oxide
- Di (Trialkyl lead) oxide plus an alkyl hydroxide to produce a trialkyl lead alkoxide
- Trialkyl lead chloride plus dialkyl hydrogen phosphide to produce trialkyl lead dialkyl phosphide
- Trialkyl lead hydroxide plus hydrogen azide to produce trialkyl lead azide.

Although the following lead organic compounds have been produced, no explicit current manufacturers were identified: tributyl lead acetate, triethyl lead acetate, and triphenyl lead acetate, used as fungicidal agents in paints; alkyl lead compounds for use as anti-mildewing agents in the cotton industry; and mercuric chloride mixed with diethyl lead dichloride, used for seed treatment.

Although not certain, it is most likely that Houston Chemical Company has produced reactive lead alkyls and mixtures thereof for sale for the manufacture of other compounds, such as ethyl mercuric chloride from tetraethyl lead (TEL). This may explain a portion of the use of 164 tons (181 tons) of metallic lead consumed in 1975 for chemicals other than gasoline additives. The sodium-lead alloy processes would, of course, have been used for the manufacture of such TEL. The process employed by Alfa Products Division of Ventron Corporation was not established, but a very good possibility is that triphenyl aluminum is reacted with a soluble lead salt in a hydrocarbon solvent.

Capacity, production levels and other specifics about these compounds are unknown. All are estimated to be quite minor, with negligible effect upon the lead industry.

4.4 REFERENCES

American Society for Testing and Materials. 1978a Part 8. Nonferrous Metals. Standard B29-55 (Reapproved 1977). American Society for Testing and Materials, Philadelphia, Pennsylvania. pp 51.

American Society for Testing and Materials. 1978b. Part 8.Nonferrous Metals. Standard B32-76. Solder Metal. American Society for Testing and Materials, Philadelphia, Pennsylvania. pp 57.

Anderson, E.V. 1978. Phasing Lead Out of Gasoline: Hard Knocks for Lead Alkyl Producers. Chemical and Engineering News. February 6. pp 12-16.

Anonymous. 1976. World's Lead Use Rate Expected to Decline. Columbus Dispatch, Columbus, Ohio. Sept. 6.

Anonymous. 1977. Private communication from a battery manufacturer.

Battelle-Columbus. 1972. A Study to Identify Opportunities for Increased Solid Waste Utilization, NTIS PB 212730.

Battelle-Columbus. 1975. Inorganic Color Pigments (Task 22) and Inorganic White Pigments (Task 23). Draft report under Contract No. 68-02-1323 to U.S. Environmental Protection Agency, Control Systems Laboratory, Research Triangle Park, North Carolina. 101 pp.

Beltz, P. R., R. H. Cherry, P.S.K. Choi, G. W. Collings, P.W. Cover, M.S. Farkas, B.C. Kim, P.W. Lerro T.A. McLure, D.N. Williams, and E.T. Yeager, 1973. Economics of Lead Removal In Selected Industry. Battelle Memorial Institute, Columbus, Ohio. U.S. Environmental Protection Agency. pp 53-70.

Callaway, H.M., 1962. Lead, A Materials Survey. Information Circular 8083, U.S. Department of The Interior, Bureau of Mines, Washington, D.C., 194 pp.

E. I. DuPont de Nemours and Company. 1964. Regulations Governing the Handling and Blending of DuPont Lead Antiknocks for Locations Receiving Antiknock in Tank Cars or Tank Trucks. E. I. DuPont de Nemours and Company, Wilmington, Delaware. January. 13 pp.

E. I. DuPont de Nemours and Company. Undated. DuPont Antiknocks. Product Bulletin. Petroleum Chemicals Division, Wilmington, Delaware. 14 pp.

E. I. DuPont De Nemours and Company. 1978. Fall 1978 ESCON Forecast. Technical Brief No. 7812. Wilmington, Delaware. September 22. 5 pp & Attachments.

Ethyl Corporation. Undated. A Guide to the Safe Handling and Mixing of "Ethyl" Antiknock compounds. Ethyl Corporation, Houston, Texas. 19 pp.

Ethyl Corporation. 1978. Yearly Report of Gasoline Sales, by States, 1977. Petroleum Chemicals Division, Houston, Texas. pp2-12.

Ewing, B. B., and J. E. Pearson. 1974. Lead in the Environment, from Advances in Environmental Science, and Technology, Vol. 3. Editors: J. N. Pitts, and R. L. Metcalf. John Wiley & Sons, New York 126 pp.

Faoro, R. B. and T. B. McMullen. 1977. National Trends in Trace Metals in Ambient Air EPA-450/1-77-003, U.S. Environmental Protection Agency, Monitoring and Reports Branch, Research Triangle Park, North Carolina. 33 pp.

Furno, F. 1978. Personal communication from Celanese Piping Systems, Inc. Hilliard, Ohio.

Hallowell, J. B., J. F. Shea, G.R. Smithson, Jr., A. B. Tripler, and B.W. Gonser. 1973. Water Pollution Control in the Primary Nonferrous-Metals Industry, Vol. I. Copper, Zinc, and Lead Industries, Office of Research and Monitoring, U.S. Environmental Protection Agency, Washington, D. C., EPA-R2-73-247a, 177 pp.

Lutz, G. A., A.R. Levin, S. G. Bloom, K. L. Nielsen, J. L. Cross, and D. L. Morrison. 1970. Technical Intelligence, and Project Information System for the Environmental Health Service, Vol. III. Lead Model Case Study, by Battelle Memorial Institute, Columbus, Ohio for U.S. Department of Health, Education, and Welfare Environmental Health Service.

McKay, J.E. 1967. Lead.In: Kirk-Othmer Encyclopedia of Chemical Technology, 2nd Ed., Vol 12, A. Standen (ed.), Interscience Publishers, John Wiley and Sons, Inc., New York pp 207-247.

Metals Handbook. 1961. Metals Handbook. 8th Ed. Vol. 1. American Society for Metals. Cleveland, Ohio.

Modern Plastics. 1976. Heat Stabilizers. Staff Article, Modern Plastics, September. pp 43.

National Petroleum News Factbook Issue. 1978. National Petroleum News, McGraw-Hill Inc., New York, New York. June. 193 pp.

National Sanitation Founcation. 1977. Standard No. 14 for Thermoplastic Materials, Pipe, Fittings, Valves, Traps, and Joining Materials. Adopted 1965; Revised February, 1977. National Sanitation Foundation, Ann Arbor, Michigan. 86 pp.

Prescott, J. H. 1977. U.S. Oil Refiners Gird for Lead-in-Gasoline Cuts. Chemical Engineering 84: 1 66-68, January 31.

Ryan, J. P., and N.M. Hague. 1976. Mineral Facts and Problems. Lead. Bulletin 667, 1975 Edition. Bureau of Mines, U. S. Department of Interior, Washington, D.C. pp. 591-610.

Shapiro, H., and F. W. Frey. 1968. The Organic Compounds of Lead. In: Kirk-Othmer Encyclopedia of Chemical Technology, 2nd Edition, Vol. 12. A Standen (ed), Interscience Publishers, John Wiley and Sons, Inc., New York, New York. pp 282-299.

Shelton, E.M. Motor gasolines. Semi-Annual Summer and Winter Surveys, 1965-1977. U. S. Department of Energy, Bartlesville Energy Technology Center, Bartlesville, Oklahoma.

Short and Associates. 1976. Preliminary Technological Feasibility, Cost of Compliance, and Economic Impact Analysis of the Proposed OSHA Standard for Lead. Prepared for OSHA, U. S. Department of Labor, by J. Short and Associates, Salt Lake City, Utah, 327 pp.

Sobotka. 1976. Economic Impact on Petroleum Refineries of Lead Additive Phasedown. Final Report to U. S. Environmental Protection Agency Contract No. 68-01-4174, Sobtka and Company, Inc., Montpelier, Vermont, p 71.

U. S. Bureau of Mines. 1975. Lead. Preprint from the 1974 Minerals Yearbook. U. S. Department of the Interior, Washington, D. C. 26 pp.

U. S. Bureau of Mines, 1976. Lead. Preprint from the 1975 Minerals Yearbook. U. S. Department of The Interior, Washington, D. C. 28 pp.

U. S. Bureau of Mines, 1977. Commodity Data Summaries, 1976. Lead. U. S. Department of The Interior, Washington, D. C.

U. S. Bureau of Mines. 1978a. Lead Industry in March, 1978. Mineral Industry Surveys, U. S. Department of The Interior, Washington, D. C. 7 pp.

U. S. Bureau of Mines. 1978b. Lead. Mineral Commodity Summaries. U. S. Department of The Interior, Washington, D. C. pp 90-91.

U. S. Bureau of Mines. 1978c. Personal communitation from J. Rathjen to R. W. Hale.

U. S. Department of Agriculture. 1975. The Pesticides Review. Agriculture Stabilization and Conservation Service, Washington, D. C. 58 pp.

U. S. Department of Commerce. 1973. Bureau of Census, 1966-1973, U. S. Department of Commerce, Washington, D. C.

U. S. Department of Labor. 1978. Occupational Exposure to Lead. Final Standard. Occupational Safetv and Health Administration, Washington D.C. 43 FR 52952-53014. November 14.

U. S. Environmental Protection Agency, 1973. Part 80. Regulation of Fuels and Fuel Additives; Control of Lead Additives in Gasoline. 38 FR 33724-33711. December 6.

U. S. Environmental Protection Agency, 1975 a. Economic Impact of Proposed Water Pollution Controls on the Nonferrous Metals Manufacturing Industry, Phase II, EPA 230/1-75-041 p/III-4, U. S. Environmental Protection Agency, Washington, D. C. March. Various pagination.

U. S. Environmental Protection Agency. 1975b. Development Document for Interior Final Effluent Limitations guidelines and Proposed New Source Performance Standards for The Lead Segment of The Nonferrous Metals Manufacturing Point Source Category. EPA 440/1-75-032 a. February. pp 124.

U. S. Environmental Protection Agency. 1976. Regulation of Fuels and Fuel Additives; Control of Lead Additives in Gasoline. 41FR 42675-42677 Sept. 28.

U. S. Environmental Protection Agency. 1977a. Control Techniques for Lead Air Emissions. 2 Vols. EPA-450/2-77-012. Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. Various pagination.

U. S. Environmental Protection Agency. 1977b, Development Document for Effluent Limitations guidelines and New Source Performance Standards for the Miscellaneous Nonferrous Metals Segment of The Nonferrous Metals Point Source Category. EPA-440/1-76/067. March. pp. 40-44.

U. S. Environmental Protection Agency. 1978. Pesticides Branch. Personal communication to R. Ewing.

U. S. Tariff Commission. 1968. Summaries of Trade and Tariff Information, Schedule 4, Vol. 10, U. S. Tariff Commission, Washington, D. C.

Walker, P. 1977. Maintenance - Free Battery Poses Problems for Secondary Plants, Am. Metal Market, June 16.

Walsh, T. 1977. Maintenance - Free Battery Seen Changing The Industry. Am. Metal Market, June 15.

Weinberg, E. L. 1976. Heat Stabilizers. 1975-1976. Modern Plastics Encyclopedia. McGraw-Hill, Inc., New York, New York. pp 208-209.

World Metal Statistics. 1977. Principal End Uses of Lead. 30 (12) 73-74. World Bureau of Metal Statistics, London, England.

Wright, J. A. and R. B. Kubalak. 1976. Lead/Zinc Industry Update - 1976, Mining Congress Journal, 62(11), p 44-46.

Ziegfeld, R. L. 1964. The Importance and Uses of Lead, Arch. Env. Health, 8 Feb.

5.0 INTRODUCTION OF LEAD INTO THE ENVIRONMENT

5.1 SUMMARY

No complete material balance appears to have ever been made for lead, perhaps because of the unquantifiable nature of some of the emissions which proceed at an imperceptible rate from the enormous inventory of lead as a "resource in use". Estimated air emissions are probably the most completely identified and quantified. The recent EPA report (U.S. Environmental Protection Agency, 1977a) covers most of the principal types of emission sources, for which estimates are provided.

There appears to be no comparable inventory of water emissions. Part of the problem here is that much, if not most, of the lead entering the hydrosphere was originally an air emission, and double-counting is a problem not amenable to an easy solution. Efforts are made in this section to identify the significant sources of lead entering the waters of the United States each year; the probable contributions of predecessor air emissions to these flows are noted and estimated where a basis for estimation exists. The estimation of solid waste disposed to landfills as solid waste from a mineral processing or manufacturing operation is fairly straight-forward; however, data are fragmentary on other more dispersed or unmeasured streams.

Existing information is summarized on the types and modes of dispersion, the receiving media (air, water, and/or land), estimates of the amounts of lead released, and when available, information on the impacts of such releases. The available or derived estimates are given in Table 5.1 along with comments as appropriate.

As a result of the widespread use of lead and its compounds described in Chapter 4, and the losses to the environment associated with that use, lead is detectable at above natural levels in all three compartments of the environment--air, water, and soil. Concentrations of lead in the environment and their effects on human intake are discussed in Chapter 8.

5.2 SOURCES

5.2.1 Lead Producing Industry

5.2.1.1 Primary Lead--

5.2.1.1.1 Mining and smelting lead ores--The mining and milling of lead ores results in the release of lead to all three media with the largest quantity of lead, in mill tailings, disposed to the land.

TABLE 5.1. ESTIMATED RELEASES OF LEAD TO THE ENVIRONMENT[a,b]

Source	Metric tons/year To the Atmosphere	To Wastewater	To the Land
Lead Producing Industry			
Primary lead	893	200-500	21,000
Primary zinc	112	3.7	1,010
Primary copper	1,314	12.5	1,590
Secondary lead	750	Sewered	3,260
Secondary brass and bronze	47	10	460
Lead Consuming Industries			
Battery manufacture	82	1-340	40
Lead alkyl manufacture	1,000	60	None
Lead oxide & Pigments	112	Sewered	200
Lead stabilizers	40	Sewered	40
Cable covering	113	Minor	Minor
Type metal	435	Minor	Minor
Can Soldering	60	Unknown	Minor
Ceramics	600	Unknown	Minor
Metallic lead products	77	Minor	Minor
Indirect Sources			
Gasoline distribution	420	None	None
Gasoline combustion	122,100	Indeterminate	Final Sink
Waste oil disposal	3,400	Indeterminate	4,650
Coal combustion	225	Minor	4,275
Oil combustion	100	Minor	In Ash
Cement manufacture	312	Minor	Unknown
Iron and steel manufacture	605	Unknown	Unknown
Grey iron products	1,080	Unknown	Unknown
Ferro alloy production	30	Unknown	Minor
Solid Waste incineration	1,170	Unkown	In Ash
Sludge disposal	5	Unknown	2,400

[a] Source: U.S. Environmental Protection Agency (1977a); Battelle-Columbus estimates.

[b] Basis: 1975 data.

The emissions to the air from the mining and milling of lead ores are principally in the form of particulates emitted during blasting, transport, crushing, and grinding of the ores. The largest portion of lead production (80 percent) comes from underground mines in southeastern Missouri. The particulate emissions from mining are those contained in the ventilation air circulated through these mines, which are usually wet, so that no general dusting is encountered within the mine (Hallowell, 1970). In open-pit mines, associated with some mixed ores (i.e., Pb-Cu, Cu-Pb-Zn ores) the dust from mining operations is more obvious in terms of blasting and loading operations. Additional fugitive dust is generated during hauling or transport by truck or conveyors from mine to mill, at transfer points and during crushing, screening and dry grinding operations. Most dusty operations are hooded and the dust is collected and returned to the process stream. Other milling operations (e.g., wet grinding) are not a source of dust.

All the above emissions consist of lead ore particles-basically insoluble lead sulfides--relatively coarse in particle size, and fall to earth within a short distance of the point of generation. The net emission of lead to the atmosphere from the crushing and grinding of lead ores is estimated to amount to 81 metric tons of contained lead annually. However, lead is also recovered from copper ores (mined in very large tonnages, generally in open-pit mines under dry conditions) and from copper-lead-zinc ores, also mined in large tonnages. Crushing and grinding of these other ores is estimated to release an additional 412 metric tons, for a total annual lead emission of 493 metric tons. (U.S. Environmental Protection Agency, 1977a).

Losses of lead to the water from lead ore mining and milling operations vary according to lead contents of mine drainage and mill tailings pond overflows. These two modes of release of lead are highly dependent on the individual circumstances of the amount of water present in a mine, and the climate (i.e., rainfall) in which the tailings pond operates. The amount of mine drainage generated by a mine depends on the specific geology of an ore deposit, i.e., whether the water in the mine is or becomes acid or alkaline either from its instrinsic nature or after interaction with exposed strata and ore. The Bonneterre Formation in which the southeastern Missouri mines operate is a productive aquifer, and most mines must pump 18.9 to 26.5 cu m/min (5,000 to 7,000 gpm) to prevent the mines from flooding (Jennett and Wixson, 1972).

The milling or concentrating of ore results in the separation of a concentrate from a gangue, which is transported as a slurry to a tailings pond for disposal on land. The local climate (i.e., rainfall) along with water-use practice in the mill, determine whether an excess of water is discharged from the tailings pond. Thus, any estimate of the amount of lead entering surface waters from lead ore mining and milling operations is affected by the highly individual circumstances of mine drainage quantity and chemistry and the climate and water use practices of the milling operations. Additionally, the estimate of lead losses to water from the mining and milling of lead ores is further subject to judgments concerning the category of ore included, in that lead is extracted not only from lead ores but also from other ores containing lead, e.g., lead-zinc ores and copper-lead-zinc ores.

One approach to estimating the lead loss to surface waters from lead ore mining and milling operations is the derivation of a "unit waste load" on the basis of measured flows and concentrations. Some example values of concentrations and flows from lead mining and milling operations are listed below (U.S. Environmental Protection Agency, 1975b):

Flow,	Plant A	Plant B	Plant C
cubic meters/day	8,300	29,000	1,426
gallons/day	2,190,000	7,700,000	377,280
Lead Concentration, mg/1	0.05	0.093	<0.024
Unit Waste Load, kg Pb/metric ton of ore	0.00012	0.071	0.013
Average Unit Waste Load, kg Pb/metric ton of ore		0.028	

The flow, concentrations, and unit waste loads given above reflect total discharge from mine and mill operations. Note the wide variation in flows and resulting derived unit waste loads. While this approach is far from rigorous, aggregated data on total flows are not available, and the estimation is necessarily based on tonnages, which are recorded.

The classes of lead-containing ores to which the unit waste load may be applied and their 1975 tonnages are (U.S. Bureau of Mines, 1976):

Class of Ore	Metric Tons	Percent of Total Lead Production
Lead	7,855,300	86
Zinc	5,657,800	1
Lead-Zinc	2,383,800	10
Cu-Pb,Cu-Zn, Cu-Pb-Zn	2,143,200	2
All Other	37,952,000	1
	55,992,100	

If the "unit waste load" derived as described above is applied to only lead ores, the estimated annual loss of lead to water would be approximately 220 metric tons. Extending this factor to the other lead-copper-zinc ore combinations would increase the estimate to about 500 metric tons/yr. Extending this to the "all other" category of ores is less appropriate. Over 98 per cent of these, primarily copper ores, are mined and milled in Arizona in net evaporation areas, and water discharges are minimal. For the purposes of this estimate they can be neglected.

Another approach to estimating losses of lead to the water from mining and milling operations utilizes the recently-promulgated Effluent Limitations Guidelines for Base and Precious Metals (U.S. Environmental Protection Agency, 1978a). The effluent limitations are expressed in concentrations rather than in units of production because it was not possible to develop an easily applicable relationship between units of production and waste water discharged by mines and mills in this category. Allowable lead discharge limits, based on best practicable technology (BPT), are 0.3 mg/1 (30-day average).

The original guidelines development document (U.S. Environmental Protection Agency, 1975b) modeled the lead mining and milling industry in terms of representative mines and mills. Using this approach to estimating produces estimated lead contents of wastewater discharges of 30 metric tons in 1977. BAT (Best Available Technology) and NSPS (New Source Performance Standards) regulations are to be proposed in 1979, which will further reduce allowable discharges.

The solid wastes from milling of lead ores contain some residual lead content since the recovery process (usually flotation) is not 100 percent efficient. The lead content in concentrator tailings from various major U.S. operations has been reported to range from 0.09 to 0.25 weight percent (AIME, 1970). The quantity of tailings may be taken as 93 to 95 percent of the ore mined. These factors lead to an estimate of 18,000 metric tons per year of contained lead being disposed of in the tailings from lead and major classes of mixed ores (i.e., Pb, Pb-Zn, Cu-Pb-Zn). This lead is principally in its initial galena form (lead sulfide). The potential for mobilization of this lead would include the possible paths of the tailings pond overflow, seepage into the underlying strata, and weathering by wind and runoff.

The smelting of lead ores results in emissions of fume and particulate to the atmosphere, the generation of lead-containing wastewaters, and the generation of slags and sludges which are disposed of to the land. The emissions of lead to the atmosphere from primary lead smelting include emissions of lead-containing fume and particulate from the pyrometallurgical unit processes of sintering, the blast furnace, and the dross reverberatory furnace. In addition, fugitive emissions are generated from concentrate handling, storage, and transfer, and leaks from ducts and furnace openings. The estimated net emissions of lead to the atmosphere from primary lead smelting operations are reported as 400 metric tons per year (U.S. Environmental Protection Agency, 1977a). These emissions would include those stemming from fume i.e., lead oxide particles of small size.

In a survey of water pollution control in the primary nonferrous metals industry made for EPA by Battelle's Columbus Laboratories (Hallowell, et al., 1973), the effluents from the tailings ponds were reported to contain anywhere from no lead to 800 μg/l; these streams originally contained from 0 to 50 μg/l as measured at points considered unaffected by plant effluents.

As part of the development of effluent limitations, the flows and lead concentrations of all lead smelter wastewaters were determined in 1973 (U.S. Environmental Protection Agency, 1975c). The estimated 1975 net release of contained lead, based on those measurements, was 2.5 metric tons.

A recent survey for the Office of Solid Waste Management idenfified the solid wastes generated by the primary lead smelting industry as slags, drosses, and sludges. On the basis of average analyses, the contained lead content of these wastes was reported as 2,640 metric tons. The lead in these wastes was mostly in the form of slags with an unknown but probably slow rate of release. Some lead was in the form of wastewater treatment sludges (i.e., most likely the hydroxide form). These wastes were all discharged to the land on the smelter sites. (Calspan, 1977).

5.2.1.1.2 Mining and smelting of other lead-containing ores--Lead occurs in varying amounts in many metal ore deposits and consequently travels with the other materials during processing. Depending on the amount of lead present and the particular process, the lead may be recovered as a by-product or appear in the wastes and residues from various processes. The residues, depending on their mode of formation, may be transferred between industries if their content of metal values makes this economically advantageous. Examples of residues which have been traditionally transferred between industries are lead-containing flue dusts from the zinc and copper industries to the lead industry; solution purification residues (lead cake) from the zinc to the lead industry, and zinc-containing slags from the lead industry to the zinc industry. (In some cases, slags from lead blast-furnaces are fumed at the lead plant to produce zinc oxide as a co-product of lead.) Several industries in this category produce noteworthy releases of lead to the environment. Unfortunately, no consistent data base exists for releases of lead to various media for all of these industries. Estimates of lead releases from processing these ores are presented below to the extent that available information allows.

As with lead ore the handling of other ores in processing results in the generation of dusts generally consisting of relatively coarse particles. As noted previously, these types of emissions are considered as fugitive; being coarse, the particles settle rapidly and are less respirable and produce more localized impacts (i.e., confined to the area of the operations) relative to other process emissions from thermal processes, high stacks, or mobile sources.

The lead intrinsic to these other ores follows similar paths to water and land as described for lead milling and tailings disposal. Depending on the lead content of the ore and the nature of the process, lead will appear in the process water discharge from the ore processing steps (e.g., in flotation tailings pond overflow), and in the waste solids disposed of in tailings ponds, as well as in dusts and runoff produced by weathering of the tailings deposit, to the extent that lead is present in the ore or tailings. (U.S. Environmental Protection Agency, 1976a).

The presence of lead in almost all mine and mill process water streams and in discharge streams is indicated by the data obtained during a survey of the metal ore mining and milling industry (U.S. Environmental Protection Agency, 1975b). Examples of the data obtained are listed below:

Type of Mine or Mill	Emission Source	Lead Concentration, mg/1
Iron	Mine	0.023
Iron	Mill	0.111
Copper	Process	<0.05-0.40
Lead-Zinc	Mine, Mill, Process	0.05-0.25
Silver	Process	<0.1
Aluminum	Process	0.10
Molyldenum	Discharge	0.03
Copper-Molydenum	Discharge	0.02
Uranium	Discharge	0.1
Platinum	Process	0.01

Although lead is present in many of the process streams, the amount discharged comprises only a few tons, based on the estimated concentrations and actual reported discharge flows. Many metal mining operations are in the western desert and produce no discharge of water. Others in areas of net water accumulation either do or are beginning to practice water recycle. Thus, while lead is present in most mineral processing streams, concentrations in actual discharge streams are relatively low, i.e., around the drinking water level of 0.05 mg/l. These discharges still represent concentrations above the levels generally encountered in surface waters. Thus, metal ore mining and milling operations may be characterized as diffusely scattered sources of low-level contributions which generally raise the level of lead concentrations in the receiving waters.

The contributions of lead to the environment from pyrometallurgical processes is most significant in terms of emissions to the atmosphere from a few high temperature processes, the content of lead in some wastewaters, and in some metallurgical wastes disposed of to the land. Notable lead emissions to the atmosphere are associated with the smelting processes for copper and zinc, and have been estimated to amount to 1,314 and 112 metric tons, respectively, of contained lead for the year 1975. (U.S. Environmental Protection Agency, 1977a).

Lead was present in the wastewaters from electrolytic refining operations in the copper industry in a sufficient number of cases to allow the development of analytical, flow, and unit waste load data. It is estimated that 12.5 metric tons of contained lead was discharged by those operations in the year (1973) the data were developed (U.S. Environmental Agency, 1975d). A study of wastes destined for land disposal from copper smelting and refining operations provided an estimate of 1,590 metric tons of lead as being contained in all process wastes such as slags, flue dusts, sludges and other process wastes. (Calspan, 1975).

Lead in the wastewaters discharged by zinc smelters was found to range in concentration from 0.02 to 1.35 mg/l (U.S. Environmental Protection Agency, 1975a). Using data gathered on the concentrations and flows for five individual smelters, the total release of lead in the wastewaters is calculated to have been approximately 3.7 metric tons for 1973.

The study of wastes generated by the primary zinc industry which are deposited on the land (slags, dusts, sludges, and water treatment residues) resulted in an estimate of 1,010 metric tons of contained lead. These wastes were almost all disposed of on the plant site where they were generated (Calspan, 1975).

5.2.1.2 Secondary Lead--

Secondary lead smelting is considered to include both the recovery of storage batteries (the major segment of the secondary lead industry) and various other processes which recover lead and lead alloys. The principal operation of scrap battery smelting is the blast furnace, while recovery of other forms of lead involve rotary, reverberatory or other forms of furnaces.

Because of the diversity and lower grades of the feeds and operations of the smaller segments of the industry, and a probably lower level of effective pollution control, the estimated emissions of lead to the atmosphere (750 metric tons) (U.S. Environmental Protection Agency, 1977a) and the amounts of lead wasted to land disposal (3,260 metric tons) (Calspan, 1975) exceed those of the primary lead smelting industry. There was not discovered any reasonable basis for estimation of losses of lead from this industry to wastewaters. In general, it may be observed that storage battery recovery includes the step of "battery breaking" in which the nonmetallic (rubber or ABS plastic) case is separated from the inner assembly of plates and separators. The battery-breaking operation obviously results in the liberation of acid and sludge from the battery. Battery breaking is sometimes done on the battery smelting plant site, sometimes as part of a diverse scrap metal operation, and occasionally as the sole activity of a small independent company. In cases of integrated battery smelting operations, the battery breaking operation is acknowledged as a generator of acid wastes, and special concrete aprons and runoff collection and treatment are provided. It is estimated that the majority of scrap operations are sources of acid runoff and drainage which is saturated with lead. These operations constitute a diffuse and unassessed segment of industrial operations. They are estimated to account for a portion of the release of lead in the flows of sewage and drainage from municipal-industrial complexes.

The sweating process (See section 4.2.2.1) gives rise to both gaseous and particulate atmospheric emissions. The gaseous portion consists of sulfur oxides, nitrogen oxides, fuel combustion products, and air. The particulates are unburned fuel, fumes, dusts, fly ash, and soot. These emissions are controlled by baghouses, with scrubbers used in some plants. Solid wastes from this process are the nonmetallics and other metallic residues remaining after lead removal (U.S. Environmental Protection Agency, 1977a).

Both atmospheric emissions and solid wastes result from the reverberatory sweating process. Gases and particulate matter containing fumes and dusts are the atmospheric emissions. The gases consist of combustion products, sulfur oxides, unburned fuel, and hydrocarbons pyrolyzed from the organic compounds mixed with the scrap. Lead, additive metals used to alloy lead, antimony, and nonmetallic materials such as fluxes or organic contaminants from the scrap are contained in the fumes and dusts. The atmospheric emissions are controlled by use of a baghouse, scrubber, or both. There also may be settling chambers or gas cooling devices. Wet lime scrubbers are used in some plants to control sulfur oxide emissions.

5.2.1 Secondary Brass and Bronze

Lead is encountered in the brass and bronze industry, which to a large extent coincides with the secondary copper industry, principally in the forms of solder residue on recycled automobile radiators, and as deliberate additions of up to 1 percent of lead to produce leaded (free-machining) brasses and as deliberate additions of 10 to 20 percent by weight in specialty alloys such as the red brasses. Emissions of lead to the atmosphere (estimated at 47 metric tons per year) result from secondary copper blast furnace and reverberatory smelting and melting, refining, and alloying furnaces (U.S. Environmental Protection Agency, 1977a). Lead in wastewaters from secondary copper operations results from operations such as slag quenching, wet-grinding of residues, and wet air-pollution control measures. The total loss of lead to the wastewaters by the secondary copper industry was estimated from (1973) data developed during effluent limitations guidelines studies as amounting to 10 metric tons (U.S. Environmental Protection Agency, 1975e). Another EPA study on land-disposed wastes from the industry developed an estimate of 460 tons of lead contained in slags and sludges from the secondary copper smelting industry (Calspan, 1975).

These lead emissions and wastes are generated largely in metropolitan industrial areas, with many wastewater discharges being routed to municipal sewer systems, and land-destined wastes being disposed of partly on the plant site and partly to municipal landfills.

5.2.2 Lead Consuming Industry

5.2.2.1 Storage Battery Manufacture--

Storage battery manufacturing operations result in annual losses of lead to the air estimated at 82 metric tons (U.S. Environmental Protection Agency, 1977a), 1 to 340 metric tons in wastewaters (Versar, 1975) and 39 metric tons of contained lead in wastes destined for land disposal (Crandall and Rodenberg, 1974). The emissions of lead to the atmosphere arise from melting, casting, oxide production operations, and the grinding and handling of dry metal and oxide powders prior to their formation into a paste. The pasting operations can be a source of air pollution because of the handling of finely divided lead oxide (<50 microns). These operations are carried out under well ventilated hoods, with the hood effluent being passed through towers and washed with water to remove particulate matter which goes into a sump.

The wide range in the estimate of losses of lead in wastewaters is due to two factors: differences between two current manufacturing processes and the wide range of wastewater management practices such as recycle and nature of chemical treatment (if used). Plants making "dry-charge" batteries actually fill batteries with acid, charge the battery, and drain the acid, resulting in a larger amount of waste acid. Plants making "wet" batteries produce lead-containing acid wastes only from spills, battery-washing, and general plant washing and sump overflows from the paste-making operations. The wastes destined for land disposal include water-pollution-control sludges, and miscellaneous scrap and plant wastes.

5.2.2.2 Gasoline Antiknock Additives--

Lead is introduced into the environment at three points in the lead alkyl cycle: from its manufacture; from the distribution and transfers of leaded gasoline on its way to the consumer; and finally, when it is burned in an internal combustion engine.

5.2.2.2.1 Manufacture--The manufacture of lead-containing antiknock compounds involves emissions to the atmosphere (1975) of an estimated 1,000 metric tons of contained lead (Versar, 1977). These losses are principally in the form of the processed organic compounds and fume and lead oxide particulates generated by the furnaces used to recover lead from process sludges. These emissions occur only at the six antiknock manufacturing sites.

The losses of lead in wastewaters from lead alkyl manufacturing plants was estimated at 60 metric tons (for the year 1975) on the basis of discharge information obtained from three plants. A recent study for the Office of Solid Waste Management Programs established that no process wastes were disposed of to the land by these plants on a regular basis; that is, the only lead-containing waste was the process sludge, which is reclaimed at all plants (Processes Research, 1976).

The losses to the air and water are judged to be directly related to production rates and will thus change with any change in production level.

5.2.2.2.2 Gasoline distribution--Most of the lead discharged into the atmosphere as a result of the use of lead alkyls as antiknock agents is, of course, discharged in the exhaust gases from interal combustion engines. As noted earlier (Table 4.16), the quantity of lead used in lead alkyl antiknock fluid in 1977 was estimated to be approximately 155,000 metric tons. All of this is presumed to eventually be dissipated, most of it in the exhaust gases. However, losses also occur in the distribution chain leading to the vehicle gasoline tank; at the refinery, at the transfer terminal, at the bulk station, and finally, at the service station.

It should be noted that these losses of lead in antiknock compounds prior to combustion are not necessarily additive to lead losses generally estimated as being due to the use of leaded gasolines, since usual estimates are based on total leaded gas production rather than on consumption, and, hence, losses occurring before the actual combustion are included in the total loss.

During the filling of tanks containing gasoline, including terminal and bulk station tanks, as well as service station tanks and automobile gasoline tanks, hydrocarbon-rich air is displaced by liquid. Sheppard, et. al, (1976) estimated that this could release to the atmosphere as much as 0.2 percent by weight (∿5.6 g/gal) each time the gasoline is transferred. Similar estimates for gasoline emission factors for vehicle fueling (4.5 g/gal) have been made by EPA (U.S. Environmental Protection Agency, 1973d) as cited by Dougherty, et al., (1977) who estimated 5.3 g/gal. At the 1977 average of 7,178,000 barrels of gasoline per day (U.S. Department of Energy, 1978), this would represent a loss of approximately 200 million gallons of hydrocarbons per year. Since only about 72 percent of gasoline in 1977 was leaded (U.S. Department of Energy, 1978), at an estimated average lead content of 1.42 g/gal (Table

4.16), estimated loss of lead alkyl (as lead) was approximately 200 metric tons per year. To this must be added the gasoline vapors displaced from the underground tanks when fuel is delivered to the station, estimated to also have about the same emission factor, which would double the loss to 400 metric tons per year. However, this is a maximum loss, since, as noted in the 1972 NAS report (National Academy of Sciences, 1972), lead alkyls are less volatile than gasoline, and tend to remain behind when gasoline vaporizes.

There are also losses in transfer and storage all the way back to the refinery. The annual emission losses for floating roof tanks, which are specified for gasoline storage by current EPA regulations, are estimated as follows:

Barrels	
Tank Size	Annual Emission Losses [a,b]
50,000	117
100,000	180
150,000	269

---

[a] Source: Sheppard, et al., (1976).
[b] Estimates based on methods in API Bulletin 2517.

In anticipation of the New Source Performance Standard, which became effective March, 1974, it was assumed that, starting in 1971 all new tanks were floating roof. It was also assumed that currently 90 percent of all gasoline tanks have floating roofs. Total petroleum industry capacity for gasoline storage was taken as approximately 210,000,000 barrels, as of September 30, 1973, (National Petroleum Council, 1974). In the absence of specific data, a 50,000-barrel storage tank was assumed to be standard, i.e., the population was approximately 4,200 storage tanks. At the estimated annual loss of 117 barrels (4,914 gallons) per tank, the total loss of hydrocarbons is put at approximately 20,640,000 gallons/year. Assuming that approximately the same storage capacity was utilized in 1977, and that 72 percent was leaded gasoline containing 1.42 g/gal of lead, a total lead loss of about 20 metric tons per year is estimated.

Projecting losses of lead alkyls in the transfer and distribution of gasoline to 1980 and 1985 involves the use of a number of assumptions which may or may not be applicable, since the entire situation is presently very fluid.

Total car mileage is projected to increase, more or less proportionally to population growth and long-term secular trends. Gasoline consumption patterns are less well defined. Sobotka (1976) estimated 1985 motor gasoline consumption of 111.4 billion gallons/yr. A 1976 EPA estimate quoted by Sobotka (1976) was that highway use of gasoline (97 percent of the total) would peak in 1980 at about 107.3 billion gal/yr, producing a total use of 110.6 billion gal/yr. However none of these estimates appear to take into account the present life style in the United States as witnessed by the fact that average 1976 gasoline consumption was 107.0 billion gallons, which increased in 1977 to about 110 billion gallons (U.S. Department of Energy, 1978); consumption in 1978 probably already exceeded all of the above estimates (Table 4.16).

A more realistic forecast appears to be that provided by the ESCON model (E.I. DuPont de Nemours and Co., Inc. 1978). In this forecast, total gasoline consumption is estimated to peak in 1980 at about 115 billion gallons per year, gradually declining to about 107 billion gal/yr by 1985 and to 99 billion by 1990. According to this same forecast, leaded gasoline consumption will drop to 57.3 billion gallons in 1980, to 26 billion in 1985 and to 14.7 billion in 1990. With an assumed uncontrolled average loss rate of 5.1 g/gal, hydrocarbon losses would be a little over 210 million gallons per year in 1980, decreasing to about 195 million gal/yr in 1985. At the mandated average gasoline pool lead content of 0.59 g/gal, annual losses of lead would be about 125 and 115 tons/yr, respectively.

The rapid decline in the use of leaded gas will cause a major decline in the loss of lead alkyls in the distribution of leaded gas. Reinforcing this trend will be EPA regulations on vapor recovery, designed to control hydrocarbon emissions, which are now in the proposed rule stage. The proposed regulations have a dual thrust. Stage I regulations will require the recovery of 90 percent of the hydrocarbon vapors displaced from gasoline storage tanks above a small size (varying from 250 to 2000 gallons) during refilling, in 16 air quality control regions (AQCR's). In addition, the regulations will require vapor laden delivery vehicles to refill only at facilities equipped with such a vapor recovery system.

Proposed Stage II regulations (U.S. Environmental Protection Agency, 1976b) call for emissions during vehicle filling in 13 of these same AQCR's not to exceed a specified value, not yet finalized but expected to be within the range of 0.4 to 0.6 g/gallon of fuel dispensed. No actual regulations have yet been promulgated and the ultimate form these regulations will take is unclear. An exemption from vapor recovery requirements for retail outlets owned by an independent small business marketers having monthly sales of less than 50,000 gallons was written into (Sec. 325) the 1977 Amendments to the Clean Air Act (P.L. 95-95), which further complicates forecasting ultimate emission from vehicle fueling.

Spills at filling stations are another source of loss of gasoline and lead alkyls. Being of a random nature, they are even more difficult to quantify than the vapor losses discussed above. It is estimated that spillage is less than vapor losses, and might approximate no more than 0.1 percent loss (3 g/gallon) dispensed. The proposed Stage II vapor recovery regulations also contain a limitation on spillage, which would be permitted to occur in up to 15 percent of all vehicle refuelings (but the quantity limits on spills are not specified).

No basis exists for an estimate of vehicle fueling losses in 1980 or 1985; it will certainly be less than the estimated uncontrolled loss of ∿100 metric tons, possibly amounting to no more than one-half.

It is not possible to assign the volume of tankage at petroleum refineries, tank farms, and bulk terminals which will in the future be dedicated to the storage of leaded gasoline. However, it is expected that the storage, and the losses, will decrease proportionally to the decrease in consumption, so that losses of lead alkyls will diminish significantly. If the decrease is proportional to average lead contents (0.5 versus 1.4 g/gal), the 1977 loss of 21 metric tons per year will decrease to only about 7 metric tons of lead per year by 1980, and continue thereafter at about that level.

Losses of leaded gasoline at service stations represents a loss of organic lead in the vapor phase, to which in previous years only a small segment of the general population, service station attendants, would have been exposed. Although the number of service stations has been decreasing in recent years, there were still approximately 176,000 in 1977 (National Petroleum News, 1978). Now, however, with the increasing trend toward self-service automobile refueling, a large segment of the general population is exposed. Percentage of self-service gasoline motorist market share has increased from about 15 percent in January, 1975 to 40 percent in December, 1977 (National Petroleum News, 1978), and is forecast to continue to increase. Since there were an estimated 114,000,000 automobiles registered in 1977 (National Petroleum News, 1978), conceivably 45 million drivers could be exposed to lead alkyl fumes while refueling their cars.

Few studies of ambient alkyl lead air concentrations around service stations appear to have been conducted. Harrison, et al., (1975) determined an organic lead level of 0.59 ug/m ^{3} six meters from a gaoline pump (See Section 5.3.2.2.1).

5.2.2.2.3 Consumption--The use of lead in gasoline antiknock fluids is a totally dissipative use as well as one which is totally dispersed; there are no regions of the United States which are free from this source of lead. However, the distribution, being a function of automobile use, is population-related and hence is concentrated in metropolitan and urban areas.

The emissions of lead from the combustion of leaded gasolines conconstitute by far the largest introduction of lead into the atmosphere, almost four times as large as all other atmospheric emissions combined. The annual downward trend after years of steady increase, but in 1977 the amount was estimated to still be nearly 155,000 metric tons. (In the peak year, 1970, consumption exceeded 220,000 tons).

Although not all of this is emitted directly to the atmosphere, the bulk of it is, as both fine (<0.5μ) and coarse (>0.5μ) particles. Habibi (1970) showed approximately 10 percent of the exhaust particles to be large "gritty" ones of 0.3 to 3 mm (300 to 3000μ) in size, which fell to the ground within a few (5 to 6) meters (16 to 20 feet) of the car. The finer particles remain suspended for longer periods, but a definite gradient with distance has been shown by numerous investigators, so that these finer particles are also concentrated near roadways. The very fine particles (<0.1μ) can remain suspended in the atmosphere for very long periods and thus can travel substantial distances from the original sources, as discussed in Section 8.2.

Vehicle age (mileage) markedly influences the nature of the emissions: these tend to be of lower quantity and smaller in size when a car is new, but both increase with mileage. Ter Haar, et al, (1972) found that the mass median diameter (MMED) of particles was about 1μ at 7,250 miles, about 2μ at 16,500 miles, and about 4μ at 21,150 miles. Also, they estimated that over the lifetime of the car, about 75 percent or more of the lead burned was exhausted, 35 percent as fine particles, and 40 percent as coarse. The remainder of the lead consumed deposited in the engine and exhaust system. Engine deposits, e.g. those on the cylinder walls, would transfer to the lubricating oil and be removed when the oil is changed. Habibi, et al., found that about 10 percent of the lead was retained in the oil and oil filter. Thus, waste oil is also an important route of dispersion of lead to the environment. Most of the remaining 15 percent unaccounted for is presumed to remain in the exhaust system.

Taking the estimate that the gasoline consumed in 1975 contained 162,800 metric tons of lead (Table 4.16) and assuming that of this quantity 75 percent was exhausted and 10 percent was transferred to the lubricating oil, calculated exhaust emissions were 122,100 metric tons, with another 16,300 metric tons contained in the lubricating oil.

In 1975, approximately 50.1 million barrels (2.1 billion gallons) of lubricating oil were sold in the United States (National Petroleum News, 1978). According to an estimate by Maugh (1976) about half of the lubricating oil was consumed in use, discarded with filter cartridges, or lost through leakage. He attributes to the Environmental Protection Agency the following estimate of the fate of the 4.5 billion liters (1.2 billion gallons) recovered:

	Billions of Liters	Millions of Gallons	Percent
Burned as fuel	2.26	600	50.0
Used in road oil and asphalt	0.76	200	16.7
Rerefined	0.42	110	9.2
Unaccounted for	1.10	290	24.1
	4.54	1200	100.0

Automotive engine oils represented only about 38 percent (797 million gals) of the total 2.1 billion gallons of lubricating base oils sold in the United States in 1975 (National Petroleum News, 1978). Except for these oils, none of the other lubricating oils are exposed to lead alkyls. Also, while lead naphthenate is added to some lubricating oils as a detergent (U.S. Environmental Protection Agency, 1977b), its use in engine oils is small enough, relative to lead pickup, to be disregarded. Thus, with respect to waste oils, only automotive engine oils are regarded as significant carriers of lead, and the following estimate considers only automotive engine oils.

The role of waste oil in the entry of lead to the environment can be estimated with the assistance of several assumptions. The assumption of Maugh that one-half of the oil added is consumed in use, lost through leakage, or discarded with the filter cartridge appears reasonable and is adopted. (The fraction discarded with the used oil filter is a contribution to solid waste). Total engine oil recovered is then 398,500,000 gallons (1,355,400 metric tons). These waste automotive lubricating oils are characterized by lead contents ranging from 800 to 11,200 ppm with an average value variously quoted as 6,000 to 10,000 ppm (0.6 to 1.0 percent) (U.S. Environmental Protection Agency, 1977a). The total lead content of the recovered oil (using an average concentration of 10,000 ppm) is estimated to be approximately 13,550 metric tons. Assuming that its disposition is distributed as estimated by Maugh, the disposition of this lead would be as follows:

	Metric Tons
Burned as fuel	6,800
Used in road oil and asphalt	2,250
Rerefined	1,250
Unaccounted for	3,250

Available information on lead emissions from waste oil burned as fuel indicates that about 50 percent is emitted with the flue gas during normal operation (U.S. Environmental Protection Agency, 1977a) providing an estimate that about 3,400 metric tons of lead are emitted. The other 3,400 tons in the oil burned as fuel is presumed to report to the ash, and is disposed of in a landfill.

It is estimated that the major portion of the lead in the waste oil rerefined would appear in the rerefiner's sludge, which is largely lagooned. There remains about 3,250 metric tons of lead in waste oil unaccounted for, with likely final fates including sewering, landfill, incineration, or wasting to surface waters. The most mobile of these quantities of lead is the approximately 3,400 tons estimated to be emitted to the atmosphere from combustion of waste oil as a fuel. The next most mobile portion would most likely be the parts of the 3,250 tons unaccounted for that are incinerated or released to surface waters. That portion released to municipal sewers would be partly abated by sewage treatment, with a portion going to sewage slude (discussed elsewhere in this report). The least mobile portions of the lead in waste oils would be those in landfilled ash, road surfacing, or that directly disposed of to the land.

5.2.2.3 Lead Oxides and Pigments--

The path of lead-containing pigments and lead oxide through industrial processing, compounding, consumption, and disposal is complex and not completely documented. Pigments constitute a completely dissipative use of lead as do some uses of oxides, while the use of lead oxide in batteries is largely nondissipative.

The production of the oxides, i.e., litharge (PbO), red lead (Pb_3O_4), and lead dioxide (PbO_2) are all furnace oxidation processes and involve the collection of the product in baghouses with some potential for emissions of lead oxides to the atmosphere but little or no potential for water pollution or solid waste. Because of the value of the products and for health reasons, these emissions are generally well controlled. Annual emissions of lead to the air from oxide production in 1975 were estimated to be 100 metric tons. (U.S. Environmental Protection Agency, 1977a). Oxide production is performed by battery manufacturers, pigment producers, and lead oxide is one of the forms of reclaimed lead produced by secondary metal processors. One of the products produced by furnace oxidation is the "black" oxide which is actually a mixture of elemental lead metal particles and litharge (PbO). All black oxide and the bulk (on the order of 80 percent) of litharge (PbO) is used in battery manufacture. Some red lead (Pb_3O_4) is also used by battery manufacturers.

The losses of lead from oxide production are principally those emissions to the air, with essentially no estimated loss to the water and the losses disposed to the land being included in those general losses attributed to the secondary lead industry.

The production of basic lead sulfate (white) pigment also involves a thermal process (See Figure 4.13) with the product collected in a baghouse and handled in similar fashion to the oxides. The production processes for other lead pigments, basic lead carbonate (white lead) and the lead chromates (yellow, orange, and green), involve aqueous chemical processes with little potential for emissions to the atmosphere, but some potential for generation of wastewaters. The review of wastewater discharges from these operations included in the development of water discharge limitations for the inorganic chemicals segment of industry concluded that, with no identifiable exceptions, the pigments industry discharged all wastewaters to municipal sewer systems (U.S. Environmental Protection Agency, 1973b). In this context, insufficient data were developed to identify how many pigments manufacturers produced lead pigments (estimated as no more than a dozen in this study) or what the specific or generalized rates of discharge or contained lead might be.

A survey of wastes destined for land disposal estimated the contained lead in such wastes from pigment operations to amount to about 200 metric tons for the year 1974 (Versar, 1977).

The second largest segment of the flow of lead oxides to further compounding or consumption is the litharge consumed in the ceramics industry (on the order of 30,000 tons in 1975). Reportedly, this lead oxide is used in glazes, the preparation of which involves the mixing of the lead oxide into a water-based suspension (with or without other components), and the spraying of the mixture onto the surfaces of ceramic ware, followed by firing in an oven to produce a fused coating. The losses of lead during spraying have not been quantified, but constitute an occupational exposure. No basis is available for an estimate of the lead losses from this industry, but at least one case is known in which lead appeared in the untreated wastewaters in maximum and average concentrations of 1800 and 1160 ppm, respectively (National Commission on Water Quality, 1975). The wastewater was being treated, principally via flocculation and clarification to remove lead in the form of suspended solids, but no discharge flow or final discharge concentrations were reported.

Yet another route of consumption for lead oxides is in glass making. Leaded glass is used in television tube manufacture, making of decorative pressed and blown glassware, and in special glass used for radiation shielding. It is estimated that these types of glass account for approximately three percent of total glass production. The major release of lead to the environment from glassmaking occurs as an emission to the atmosphere during handling, weighing, charging, and melting in the glass-making furnace. This emission has been estimated to amount to 56 metric tons for the year 1975 (U.S. Environmental Protection Agency, 1977a). Releases of lead in wastewater from glass-making result principally from grinding and etching of lead glass and are estimated to amount to 0.2 to 2 metric tons per year on the basis of the information available (U.S. Environmental Protection Agency, 1974c). No basis exists for the estimation of lead contained in industrial wastes destined for land disposal. Depending on industry practices, air pollution control residues and water pollution control, sludges disposed of to the land might amount to more than the estimated releases mentioned above. Most of the leaded glass (except radiation shielding) is estimated to eventually reach municipal or industrial landfills.

Quantities of litharge (on the order of 6,000 metric tons per year) are used in the making of rubber. The oxide is added to some rubbers in proportions of approximately one to ten percent by volume to accelerate the vulcanizing reaction. Litharge is added most often to rubber serving as insulation on wire. No specific or noteworthy releases of lead to the environment are judged to be associated with this use of lead, although the use is totally dissipative. The final fate of such insulation is judged to be divided among a minor portion of recycle, some incineration, and disposal to the land.

5.2.2.4 Inorganic Lead Compounds--

The largest identifiable class of inorganic lead compounds, other than oxides and pigments, discussed in Section 5.2.2.3, includes the stabilizers used in plastics, principally in PVC polymers. Usage of lead in the plastics industry in 1976 was estimated at approximately 8,200 metric tons per year (Modern Plastics, 1976). The estimated 8,200 tons of lead so consumed would be processed first into the indicated compounds, and then be involved in the final formulation and compounding of the plastic product. In view of the lack of definitive information concerning the environmental aspects of any of the specific industrial processes involved, no basis exists for the calculation of lead losses to any media. It may be reasoned that some losses of raw or input materials occur in any industrial process, and that these generally are at least on the order of one percent of the input, and that the consumption of 8,200 tons of lead would result in at least 80 tons of losses in some form during manufacture. It could be further reasoned that since the stabilized compounds are largely produced in aqueous chemical processes, the most probable form of loss would be that to wastewaters. However, the manufacturing plants producing the compounds and the plastics mostly discharge wastewaters to municipal sewer systems where other pollutants (e.g., biological and chemical oxygen demand) are generally more significant than trace metals.

The next largest identifiable portion of lead involved in any class of compounds is an estimated 1,500 tons of lead consumed in paint driers. Reasoning similar to that above would have to be applied to these compounds in that they also undergo various compounding steps, i.e., conversion of lead to a specific compound, and formulation of the drier into paints. However, the forms of release of paint driers would include losses during the application of paints as well as losses during chemical and paint compounding.

Lesser tonnages of lead (i.e., on the order of the 133 metric tons assigned to the classification "Miscellaneous Chemicals" by the Bureau of Mines) are consumed in preparation of other listed compounds (except oxides and pigments). As noted in other portions of the text, some compounds are produced in reagent grades and small quantities for analytical use. Losses during such processing would be estimated to be proportionately smaller than for bulk processing of industrial grades of compounds, and probably are insignificant.

It can be concluded from the discussions in this and the preceding section that essentially all inorganic lead compounds, except for a portion of the oxides, are eventually dissipated through attrition or being disposed of as waste; the lead in these compounds is never recycled.

5.2.2.5 Miscellaneous Uses of Metallic Lead--

The nature of the uses of metallic lead is such that individual emission losses from their manufacture or use are modest, and they pale in comparison to the quantities of lead introduced into the biosphere through the combustion of leaded gasolines. Additionally, relatively small number of point sources is involved so that the environmental impacts are localized.

5.2.2.5.1 Cable covering and rubber hose manufacture--As described in Section 4.3.5.4, there are two types of lead use in the manufacture of electric cable; in one lead provides the final sheath, in the other it supplies the necessary restraint while a rubber base covering is vulcanized, and is then removed for recycle to the process.

The process of applying lead sheathing to preassembled electric cable involves the manipulation of lead in and around machinery so as to form a continuous envelope of lead around the cable. The sheathing may be entirely metallic or a combination of lead and plastic, depending on the degree of protection or the properties desired in the composite product. The maintenance of molten lead supplies or baths and the routing of the lead through the forming machinery result in the generation of fume, particulate, and slivers which result in not only high exposure of the machine operators but an estimated annual emission of 113 metric tons of lead to the atmosphere. Cable covering processes normally do not incorporate particulate collection devices (U.S. Environmental Protection Agency, 1977a). Because of the non-aqueous nature of the process and the available opportunity of returning scraps and wastes to the melting pot, lead in wastewaters or other forms is estimated to be zero or negligibly small.

Rubber sheathed cable and rubber hose are made in much the same fashion. A lead sheathing is applied to the assembled uncured components so that the lead sheath acts as a temporary pressure vessel or custom die to keep coils of hose under pressure during curing or valcanizing. After curing in a steam pressurized autoclave, the lead sheath is stripped and the lead recycled to the sheathing operation. Lead appears as a wastewater pollutant in the condensate from the autoclave when it is blown-down or emptied. Based on information available from effluent limitation Guidelines studies, the lead may occur in concentrations as high as 67 mg/l in the raw wastewater. Using information from the guidelines studies, it is estimated that the total potential release of lead from rubber hose manufacture is about 2.5 metric tons. This potential release would be associated with approximately 30 plant sites, about 26 of which are estimated to discharge to municipal sewers (U.S. Environmental Protection Agency, 1974d). If the plants achieved the limits of lead discharge specified by the Effluent Limitations Guidelines through pretreatment of wastes, the discharge to the sewers would be reduced to about 60 kilograms of lead per year. The 2.4 metric tons removed from the wastewater would presumably be disposed of as a sludge to a suitable landfill.

5.2.2.5.3 Can Manufacturing--Can making, actually the soldering and cleaning or smoothing of the soldered seam, involves the maintenance of solder baths, the handling of the molten metal and the wire or cloth brushing of the frozen solder seam. These operations produce emissions to the atmosphere of fume and particulate estimated to amount to 60 metric tons per year in 1975 (U.S. Environmental Protection Agency, 1977a). The potential for losses of lead in wastewater or in wastes destined for land disposal is considered to be zero or negligibly small.

However, as will be developed later in this report, the use of solder in food cans comprises one of the major sources of additions to man's body burden of lead, but is one for which alternatives are available. Though not, strictly speaking, an emission, it is one of the most important facets of the entry of lead into the environment.

5.2.2.5.4 Miscellaneous uses of metallic lead--Lead has numerous miscellaneous uses, which are generally characterized by the consumption of a relatively small tonnage, but for which lead fills a niche not easily filled by competing materials. Most of these applications utilize the high density, corrosion resistance, malleability and easy formability, or non-sparking chacter of this metal. Included are the use of architectural sheet and foil for flashing and sound proofing, and for linings of tanks and vessels for purposes of corrosion resistance in chemical process vessels (a recent noteworthy example is linings of electrostatic precipitators for cleaning corrosive industrial gas streams. Lead plate is used for spark-proof flooring in explosives plants and other hazardous areas. Lead foil, sheet and plate, and thick-walled lead containers are used for radiation shielding in nuclear applications, and around industrial and medical x-ray equipment. A high-lead glass is used for hot-cell windows. Lead is used for weights and ballast on automotive wheels, ships, rotating machinery, and as counterweights on fork-lift trucks and other machinery, and in molten metal baths as for heat treating of steel wire. In essence, there are numerous uses of lead in prefabricated and/or massive form with which there are associated no or negligible losses of lead to the environment during use, and from which the lead, being in recognizable, fairly massive form, will be largely reclaimed through recycling to the secondary lead industry.

The major release of lead to the environment associated with these uses is that occuring during the original melting and casting of the items to shape. Subsequent fabrication of rolled sheet and foil are dry mechanical processes in which releases are zero or negligibly small.

5.2.3 Indirect Sources of Lead

The sources of entry of lead to the environment described in Sections 5.2.1 and 5.2.2 have all dealt with some segment of the lead industry or with other industries in which lead products are utilized in some beneficial fashion. There are, in addition, a number of sources of lead entry into the environment where the lead is present in a process only as impurity or trace component. While most of these processes have relatively low specific emission factors, these are frequently associated with very large tonnages of process materials, with the net result that some of the emissions are significant.

5.2.3.1 Iron and Steel Production--

Releases of lead from the iron and steel industry occur due to the presence of lead in iron ore, coking coals, and in scrap which is remelted. As indicated above, even trace contaminants produce substantial emissions when multiplied by the enormous tonnages of this industry, as illustrated by estimates of 1975 production (U.S. Environmental Protection Agency, 1977a):

	Millions of Metric Tons
Grey iron	16.7
Sinter	27.9
Coke	43.8
Blast furnace	72.5
Basic oxygen furnace	65.1
Open hearth furnace	20.1
Electric arc furnace	20.6

The lead in coal appears as an emission from coke ovens with the net emitted lead content estimated as 11 metric tons for the year 1975. The lead content and emission factor is very low per ton of coal or coke, but the high tonnage of coke production results in an appreciable estimated emission of lead. Similarly, the presence of lead as a trace impurity in iron ore, combined with the very large tonnage of iron ore processed results in another appreciable estimated net emission of 18 metric tons of lead from iron ore sintering operations. These sintering operations are the first high-temperature steps in the processing of the ore and the lead is volatilized.

Similar reasoning applies to the presence of an appreciable net estimated emission of 91 metric tons of lead from blast furnaces, wherein the coke and sintered ore are processed at a successively higher temperature resulting in continued volatilization of the residual lead (U.S. Environmental Protection Agency, 1977a).

Lead is also emitted to the atmosphere from the various types of melting furnaces in the steel (basic oxygen, open hearth, and electric furnaces) and gray iron (cupola and electric furnaces) industries. These emissions are principally attributable to the presence of lead in scrap materials charged to these furnaces. Scrap makes up various percentages of the charge, ranging from 25 percent of the total iron charge up to 100 percent of the charge in some electric furnace operations. The lead emission is attributed to the presence of lead in automobile scrap (solder) and red lead paint on scrap derived from ships, bridges, structures, and to a lesser extent, other scrap containing solders, galvanizing and/or enamels. The total estimated net emissions from these scrap remelting furnaces is approximately 1,564 metric tons for the year 1975 (U.S. Environmental Protection Agency, 1977a).

While it may be reasoned that the principal source of wastewaters in primary steel operations is air pollution control, and that wastewaters or dry residues from air pollution control would thus show some lead content, there is insufficient basis for estimating the losses of lead to water and in land-destined wastes. Various individual analyses of process water streams within steel plants show lead contents above intake waters but lead is not a major pollutant controlled by any specific discharge regulation applicable to wastewaters from the steel industry. Similarly, baghouse dust from electric furnace operations (destined for land disposal) likewise show appreciable concentrations of lead, but no basis exists for an estimate of the aggregate lead released to water and land from steel melting operations. (U.S. Environmental Protection Agency, 1974a).

Lead is also an appreciable emission, estimated at 30 metric tons in 1975, to the atmosphere from the production of ferromanganese. The lead is present in manganese ore concentrate which is charged to electric furnaces for smelting (with coke). The estimated lead emission is the net lead emitted after control devices; an associated lead content is present in the dust collected by control devices which are predominantly baghouses. The dust is typically disposed of to a landfill because currently neither the manganese nor the lead content are economically recoverable. No basis exists for an estimate of the aggregate total of the lead content of these dusts which are discharged to landfills.

5.2.3.2 Cement Manufacture--

Lead is present in cement in trace concentrations and is emitted to the atmosphere during handling, transport, and especially from cement kilns. The estimated net emission of contained lead was 312 metric tons in 1975 (U.S. Environmental Protection Agency, 1977a). Review of the Guidelines Development

Document for the cement industry gave factors of about 20 percent of plants practicing dust leaching, wet slurry disposal (with possible discharge of supernatant) or wet air pollution control, all of which allow a potential release of lead to wastewater discharge (U.S. Environmental Protection Agency, 1973a). Using the unit waste load given for lead (0.99 grams per metric ton of product) for 20 percent of production results in an estimated potential release of lead in process wastewaters of approximately 14 metric tons for 68 million metric tons of production. This estimate does not include consideration of runoff flows. The highest concentration (not specified) of lead encountered was for wastewaters from a plant using oyster-shell marl for raw material.

The intrinsic lead content of the raw materials of the cement industry would be volatilized during the clinkering process in the kiln, with some lead reporting in the wastewaters of plants where water contacts the kiln dust. In other plants (an estimated 80 percent of the industry) the dusts are returned to quarries, slurried to on-site lagoons, or hauled away by contractors (presumable for land disposal). No data base is available to allow a estimation of either the amount of dust disposed of to landfills or the lead content of this dust. High alkalinity is the reason the dust is wasted, a condition which would favor the minimum solubilization of any lead present. (U.S. Environmental Protection Agency, 1973a).

5.2.3.3 Combustion of Fossil Fuels--

Lead is a trace metal impurity of highly variable occurrence in coals. Gluskoter, et al., (1977), in their exhaustive study of the occurrence and distribution of trace elements in coal, reported on the analysis of 165 U.S. coals. Lead concentration ranged from 4 to 218 ppm; with considerable differences between coals from different regions:

	Lead, ppm	
Coal Samples	Arith. mean	Geom. mean
23 Eastern Appalachian	5.9	4.7
114 Illinois Basin	32	15
28 Western	3.4	2.6

It is evident from the above that much more data would be needed than are available in order to estimate a weighted average lead content for the coal burned in a given year. Most coal currently burned comes from Eastern Appalachia and the Illinois Basin, and for purposes of calculation the two are arbitrarily averaged (geometric means) at 10 ppm.

Annual U.S. coal consumption is currently of the order of 450 million metric tons (500 million short tons), estimated (at 10 ppm) to contain about 4,500 tons of lead. On the basis of the study by Klein, et al., (1975), on the pathways of 37 trace elements through a coal-fired power plant, approximately 97.5 percent of the lead was trapped in the fly ash and the bottom ash, with only about 2.5 percent escaping. However, the plant tested was in the exemplary class, and 95 percent is estimated to be a more realistic national average. Using this latter value, lead emitted is estimated to be approximately 225 metric tons/year.

The other 95 percent of the lead, of the order of 4,275 metric tons (4,700 short tons) remains with the ash, and in larger installations, is generally disposed of to an ash pond.

Fuel oils also contain traces of lead; the EPA extimate of lead emissions (U.S. Environmental Protection Agency, 1977a) was a nominal 100 metric tons per year.

5.2.3.4 Solid Waste Incineration--

Quantitative data on the quantities of solid waste incinerated and its lead content are lacking. One estimate (U.S. Environmental Protection Agency, 1977a) was that about 1.6 kg (3.5 lb) of domestic refuse and garbage is collected per capita per day in the United States. With a U.S. population of about 210 million in 1975, total domestic solid waste is estimated to have been approximately 123 million metric tons. In 1975 an estimated 16.5 million metric tons were burned in municipal incinerators. Based on limited emission data, lead emissions were estimated at 1,170 metric tons (U.S. Environmental Protection Agency, 1977a). These emissions arise from many of the components of municipal refuse including most of the dissipative uses of lead: pigments in paints, dyes, and inks, lead in plastics, rubber, insulation, cans and other soldered components, and other forms. An important, but somewhat uncertain, quantity is the lead in waste lubricating oil from automobiles. A substantial amount of this is discarded to solid waste and finds its way to an incinerator.

5.2.3.5 Sludge Disposal--

Lead is reported present in municipal sewage sludges in the range of 10 to 26,000 parts per million, with a median concentration of 500 parts per million (Page and Chang, 1975). One EPA estimate is that 5.6 million dry metric tons (6.2 million short tons) of municipal sludge are being produced and disposed of in one manner or another each year. At the median concentration this represents about 3100 metric tons of contained lead. It may be noted that this also represents the reappearance of some of the lead emissions to water described in previous pages, with lead from used motor oil perhaps the largest contributor. The estimated general breakdown for disposal is as follows (U.S. Environmental Protection Agency, 1976c):

Method		Percent of Total Municipal Sludge	Millions of Metric Tons
Ocean Disposal		15	0.8
Incineration		35	2.0
Landfill		25	1.4
Land Application		25	1.4
(croplands)	(20)		
(other)	(5)		
		100	5.6

Ocean disposal has been favored by a number of large coastal cities. The disposal of sludge to the ocean has been shown to result in elevated levels of lead (and other metals) in bottom sediments around the point of disposal. In one study of silts, sediments and sludges in the Los Angeles and Southern California area, marine sediments affected by flows and sludges from an urbanized area show lead contents 30 or more times those in natural coastal sediments not affected by sludge or urbanization. The impact of this release has produced no notable effects in terms other than the elevated lead concentrations in the silts, i.e., no preferential concentrations in man or aquatic organisms have been noted (Young, et al., 1973). However, some heavy metals are known to bioaccumulate to where they interfere with reproduction or cause toxic effects to certain marine organisms; those of immediate concern are cadmium and mercury. Efforts are currently underway to phase-out most of the ocean dumpers and pipe dischargers by 1981 or soon thereafter (U.S. Environmental Protection Agency, 1976c).

Incineration is the most commonly used disposal option, and many incinerators are located in large urban areas. This is related in part to the fact that incineration accomplishes the greatest volume reduction (to 10 to 30 percent of the original matter dry volume) and the locations with the largest inputs of sludge are generally the areas with the least available space for disposal.

The release of lead to the atmosphere from sewage sludge incineration may be estimated on the basis of a study of selected sites performed to determine the impact of anticipated increases in incineration if ocean dumping were to be curtailed. All stack tests were on incinerators with wet scrubber controls and showed net total particulate emission rates of 0.6 to 2.4 kilograms per metric ton of charge (Battelle-Columbus, 1973). Assuming the particulates also contained the median lead concentration of 500 ppm, the 1,200 to 4,800 metric tons of particulates emitted would contain 0.6 to 2.5 metric tons of lead; a negligible quantity. The balance of the lead in the sludge would appear either in the ash or the air pollution control dust or sludge, all of which would presumably be sent to a municipal landfill.

Approximately 25 percent of municipal sludge is disposed of by land application; this disposal option has been preferred by communities with suitable available land, generally inland cities and smaller rural communities. Reportedly, trace metal concentrations may accumulate in soils so treated and some metals may produce toxic effects to plants growing in such soils. However, in actual field operations and in laboratory tests in which some metal toxicity has been observed in terms of reduced growth, lead has not been shown to be the cause of the toxicity. Interactions of lead with plants is discussed in Section 6.3 and lead in foods is discussed in Section 8.5 and 8.6.

5.4 REFERENCES

AIME. 1970. AIME World Symposium on Mining and Metallurgy of Lead and Zinc, Volume 1, Mining and Concentrating of Lead and Zinc, D.O. Rausch and B. C. Mariacher (eds.), AIME, New York, New York.

APHA, AWWA, WPCF. 1971. Standard Methods for the Examination of Water and Wastewater. 13th Ed.

Battelle-Columbus. 1973. Control Techniques for Emissions Containing As, Cd, Cu, Se, Zn, Cr, Mn, V, B, Ba, and Hg. Draft Report to U. S. Environmental Protection Agency. Columbus, Ohio.

Calspan. 1975. Assessment of Industrial Hazardous Waste Practices in The Metal Smelting and Refining Industry. Draft Report to U. S. Environmental Protection Agency. Vol. II, Primary and Secondary Nonferrous Smelting and Refining Industry. Contract No. 68-01-2604. Calspan Corporation, Buffalo, New York. April. Various pagination.

Calspan. 1977. Alternatives for Hazardous Waste Management in The Metals Smelting and Refining Industries. Draft Report to U. S. Environmental Protection Agency. Contract No. 68-01-4312. Calspan Corporation, Buffalo, New York. Various pagination.

Crandall, C. J. and J. R. Rodenberg. 1974. Waste Lead Oxide Treatment of Lead Acid Battery Manufacturing Wastewater, In: Proceedings of the 29th Industrial Waste Conference, May 7, 9, Purdue University, Lafayette, Indiana. pp 194-206.

Dougherty, M. J., A. W. Percy, E. L. Wiseman, and K. F. Lord. 1977. Gasoline Vapor Cotnrol at Service Stations. Amer. Inst. Chem. Engr. Symposium Series. 165, Vol. 73. pp 249-259.

Dougherty, M. J., A. W. Percy, E. L. Wiseman, and F. K. Lord. 1977. Gasoline Vapor Control at Service Stations. Amer. Inst. Chem. Engr. Symposium Series. 165, Vol. 73. pp 249-206.

E. I. duPont de Nemours and Co., Inc. 1978. Fall 1978 ESCON Forecast. Technical Brief No. 7812. Wilmington, Delaware. September 22. 5 pp. and attachments.

Gluskoter, H. J., R. R. Ruch, W. G. Miller, R. A. Cohill, G. B. Dreher, and J. K. Kuhn. 1977. Trace Elements in Coal: Occurrence and Distribution. EPA-600/7-77-064. U. S. Environmental Protection Agency, Research Triangle Park, North Carolina. June. 153 pp.

Habibi, K. 1970. Characterization of Particulate Lead in Vehicle Exhaust - Experimental Techniques. Environ. Sci. Technol. 4(3):239-251.

Hallowell, J. B. 1970. Trip Report on Visit to St. Joe Minerals Company Viburnum, Missouri Mine. Battelle-Columbus, 5 pp.

Hallowell, J. B., J. F. Shea, G. R. Smithson, Jr., A. B. Tripler, and B. W. Gonser, 1973. Water Pollution Control in the Primary Nonferrous-Metals Industry. EPA-R2-73-247a, Vol. I. Copper, Zinc, and Lead Industries. Office of Research and Monitoring, U. S. Environmental Protection Agency, Washington, D. C. 177 pp.

Harrison, R. M., R. Perry and D. H. Slater. 1975. The Contribution of Organic Lead Compounds to Total Lead Levels in Urban Atmospheres. In: International Symposium Proceedings, Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Vol. III. Luxembourg, Comm. of European Communities. pp 1783-1788.

Jennett, J. C., and B. G. Wixson. 1972. Problems in Lead Mining Waste Control. J. Water Poll. Contr. Fed. 44(11):2103-2110.

Klein, D. H., A. W. Andren, J. A. Carter, J. F. Emergy, C. Feldman, W. Fulkerson, W. S. Lyon, J. C. Ogle, Y. Tainai, R. I. Van Hook, and N. Bolton. 1975. Pathways of Thirty-Seven Trace Elements Through a Coal Fired Power Plant. Environ. Sci. Technol., 9(10):973-979.

Maugh, T. H. 1976. Rerefined Oil: An Option that Saves Oil, Minimizes Pollution. Science. 193:1108-1110.

Modern Plastics. 1976. Heat Stabilizers. Staff Article, Modern Plastics, September. pp 43-44.

National Academy of Sciences, Committee on Biologic Effects of Atmospheric Pollutants. 1972. Biologic Effects of Atmospheric Pollutants. Lead. Airborne Lead in Perspective. Washington, D. C., 341 pp.

National Commission on Water quality. 1975. Cost of Implementation and Capabilities of Available Technology to Comply with P.L. 92-500. Vol. III. pp. 26-4.

National Petroleum Council. 1974. Petroleum Storage Capacity. National Petroleum Council, Washington, D. C., pp 17-21.

National Petroleum News. 1978. Factbook Issue. Mid June. New York, New York.

Page, A. L. and C. C. Chang. 1975. Trace Element and Plant Nutrient Constraints of Recycling Sewage Sludges on Agricultural Land. In: Proceedings of the Second National Conference on Complete Water Reuse. Chicago, May 4-8. pp 201-209.

Processes Research. 1976. Alternatives for Hazardous Waste Management in The Organic Chemical, Pesticides, and Explosives Industries. Draft Report to U. S. Environmental Protection Agency. Contract No. 68-01-4127. Processes Research Inc., Cincinnati, Ohio. December. Various pagination.

Sheppard, W., L. Bowa, D. Hissong, D. Jenkins, R. Mendelsohn, J. Otis, C. Rodman, S. Srinivasan, R. Wise, and D. Moore. 1976. The Economic Impact of Environmental Regulations on the Petroleum Industry - Phase II Study. Battelle Memorial Institute, Columbus, Ohio. Various paging.

Sobotka. 1976. Economic Impact on Petroleum Refineries of Lead Additives Phasedown. Final Report to U. S. Environmental Protection Agency, Contract No. 68-01-4174, Sobotka and Company, Inc., Montpelier, Vermont. 71 pp.

Ter Haar, G. L., D. L. Lenane, J. N. Hu, and M. Brandt. 1972. Composition, Size, and Control of Automotive Exhaust Particles J. Air Polln. Control Assoc. 22 (1)39-45.

U. S. Bureau of Mines. 1976. Lead. Preprint from The 1974 Minerals Yearbook. U. S. Department of The Interior, Washington, D. C. 26 pp.

U. S. Department of Energy. 1978. Monthly Energy Review, DOE/EIA-0035/4, Energy Information Administration, Department of Energy, Washington, D.C. April. 10 pp.

U. S. Environmental Protection Agency. 1972. Helena Valley, Montana, Area Environmental Pollution Study. AP-91, U. S. Environmental Protection Agency, Research Triangle Park, North Carolina.

U. S. Environmental Protection Agency. 1973a. Development Document for Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Smelting and Slag Processing Segment of the Ferroalloy Manufacturing Point Source Category. EPA 440/1-73/008, Washington, D. C. August.

U. S. Environmental Protection Agency. 1973b. Development Document for Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Major Inorganics Products Segment of the Inorganic Chemicals Manufacturing Point Source Category. EPA 440/1-73/007, Washington, D. C. August.

U. S. Environmental Protection Agency. 1973c. EPA's Position on the Health Implications of Airborne Lead. U.S. Environmental Protection Agency, Washington, D. C.

U. S. Environmental Protection Agency. 1973d. Compilation of Air Pollutant Emission Factors. AP-42. Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. Various pagination.

U. S. Environmental Protection Agency. 1974a. Development Document for Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Steel Making Segment of the Iron and Steel Manufacturing Point Source Category. EPA 440/1-73/024. Washington, D. C. February.

U. S. Environmental Protection Agency. 1974b. Draft Development Document for Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Paint and Ink Formulation and Printing Point Source Category. Washington, D. C. April.

U. S. Environmental Protection Agency. 1974c. Development Document for Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Pressed and Blown Glass Segment of the Glass Manufacturing Point Source Category. EPA 440/1-74/034. Washington, D. C. August.

U. S. Environmental Protection Agency. 1974d. Draft Development Document for Effluent Limitations Guidelines and Standards of Performance - Rubber Processing Industry. U.S. Environmental Protection Agency, Washington, D. C. January.

U. S. Environmental Protection Agency. 1974e. Economic Analysis of Proposed Effluent Guidelines - The Rubber Processing Industry (Phase II). EPA-230/1-74-046. Washington, D. C. August.

U. S. Environmental Protection Agency. 1975a. Development Document for Interim Final Effluent Limitations Guidelines and Proposed New Source Performance Standards for the Zinc Segment of the Nonferrous Metals Manufacturing Point Source Category. EPA 440/1-75/032. Washington, D. C. February.

U. S. Environmental Protection Agency. 1975b. Development Document for Interim Final and Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Ore Mining and Dressing Industry Point Source Category. EPA 440/1-75/061. 2 Vols. Washington, D. C. October.

U. S. Environmental Protection Agency. 1975c. Development Document for Interim Final Effluent Limitations Guidelines and Proposed New Source Performance Standards for the Lead Segment of the Nonferrous Metals Manufacturing Point Source Category. EPA 440/1-75/032a. Washington, D.C. February.

U. S. Environmental Protection Agency. 1975d. Development Document for Interim Final Effluent Limitations Guidelines and Proposed New Source Performance Standards for the Primary Copper Smelting Subcategory and the Primary Copper Refining Subcategory of the Copper Segment of the Nonferrous Metals Manufacturing Point Source Category. EPA 440/1-75/032b. Washington, D. C. February.

U. S. Environmental Protection Agency. 1975e. Development Document for Interim Final Effluent Limitations Guidelines and Porposed New Source Performance Standards for the Secondary Copper Subcategory of the Copper Segment of the Nonferrous Metals Manufacturing Point Source Category. EPA 440/1-75/032c. Washington, D. C. February.

U. S. Environmental Protection Agency. 1976a. A Study of Waste Generation Treatment and Disposal in the Metals Mining Industry. Final Report SW-132c, EPA/OSWMP, October.

U. S. Environmental Protection Agency. 1976b. Approval and Promulgation of Independent Plans. Reproposal of Amendments to Stage II Vapor Recovery Regulations and Test Procedures and Notice of Public Hearings. Washington, D. C. 41 FR 48044-48059. November 1.

U. S. Environmental Protection Agency 1976c. Municipal Sludge Management: EPA Construction Grants Program. EPA-430/9-76-009. Office of Water Program Operations,Municipal Construction Division, Washington, D. C. April 64 pp.

U. S. Environmental Protection Agency. 1977a. Control Techniques for Lead Air Emissions. 2 Vols. EPA 450/2-77-012 . U. S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. December. Various pagination.

U. S. Environmental Protection Agency. 1977b. Air Quality Criteria for Atmospheric Lead, EPA-600/8-77-017. U. S. Environmental Protection Agency, Office of Research and Development, Washington, D. C. December. Various pagination.

U. S. Environmental Protection Agency. 1978a. Effluent Limitations Guidelines for Existing Sources, Ore Mining and Dressing Point Source Category. Final Rule. 43 FR 29771-29782. July 11.

U. S. Environmental Protection Agency. 1978b. National Primary and Secondary Air Quality Standard for Lead. 43 FR 46246-46263. October 5.

U. S. Environmental Protection Agency. 1978c. Implementation Plans for Lead National Ambient Air Quality Standard. 43 FR 46264-46271. October 5.

Versar, 1975. Assessment of Industrial Hazardous Waste Practices: Storage and Primary Battery Industries. Draft Report to U. S. Environmental Protection Agency. contract No. 68-01-2276. Versar, Inc. Springfield, Virginia. Various pagination.

Versar. 1977. Alternatives for Hazardous Waste Management in The Inorganics Chemicals Industry. Draft Report to U. S. Environmental Protection Agency. Contract No. 68-01-4190. Versar, Inc., Springfield, Virginia. January. Various pagination.

Young, C. S., T. K. Jan, and N. Rohatgi. 1973. Suspended and Dissolved Trace Metals in Wastewater Effluents. Presented at the 64th Annual Conference of the Water Pollution Control Federation, Cleveland, Ohio, September 30-October 5, 1973.

6.0 ECOLOGICAL EFFECTS

6.1 SUMMARY

Lead, at levels above "normal" has the same effect on terrestrial and aquatic organisms as most other "toxic" substances. Exposure of an individual group of organisms can not only affect those exposed, but ultimately can affect numerous other organisms, either directly through consumption of exposed organisms and the resulting exposure to lead, or by the disruption of an important activity, such as nutrient cycling, in which the exposed group of organisms is involved, and on which other organisms depend.

Lead exposure of terrestrial and aquatic protista (bacteria, protozoans, algae, fungi and viruses) can have just such an effect. Lead in various forms and concentrations has been found to be responsible for the disruption of protistan activities such as nutrient cycling, decomposition of organic matter, mobilization and biotransformation of metals and inorganic and organic compounds. A mechanism for these and other effects of lead exposure has been attributed to the alteration of normal biosynthetic pathways through direct or indirect actions on enzymes.

In higher plants, the effects of lead have been studied and found to occur rather uniformly. Lead, not considered an essential element for plants, has a variety of effects on plants; it can inhibit growth, reduce photosynthesis, interfere with cell division, inhibit enzymes unique to photosynthesis and respiration, reduce water absorption and transpiration, accelerate abcission or defoliation and pigmentation, and reduce chlorophyll and ATP synthesis. The degree or extent of lead exposure is dependent not only on the concentration and form of the lead, but also on the individual species, age of the plant, nutrient availability, soil pH, cation exchange capacity, presence of other nutrients, natural chelating agents and other factors.

There are primarily two pathways for lead to enter a plant: uptake by roots or foliage. Accumulation of lead from the soil is generally minimal, although some inhibition of root growth has been attributed to lead exposures. In aerosol form, lead can be absorbed by plant foliage; in particulate form, it can accumulate on foliage through fallout. As discussed previously, numerous factors influence the effect lead has on plants. Lichens and bryophytes, for example, are known to be capable of accumulating high levels of lead with no apparent effect. In most instances, however, a plant in good health with sufficient nutrients, moisture, temperature and light is not likely to be affected by lead concentrations of less than 100 ppm, dry weight. Perhaps the most significant effect of lead in or on plants is the potential of accumulation in higher organisms feeding on contaminated plants.

Levels of lead above the allowable drinking water standard of 50 μg/l are toxic or result in morphological changes in aquatic invertebrates. As is common in other organisms, age plays a significant role in susceptibility. Embryo and fingerling stages of fishes are more susceptible to lead poisoning than adults. Young fish may be deformed or suffer blacktail syndrome by certain levels of lead accumulation, and, in general, fish are more susceptible to poisoning in soft water than in hard water.

Waterfowl are susceptible to lead poisoning from ingestion of lead shot used by hunters. Accumulation of this nature has a significant effect on the population; an estimated two to three percent of the waterfowl in the United States die annually from this type of lead poisoning. Predatory birds may also be poisoned by eating prey containing lead shot. The deposition of ingested lead in tissues is influenced by diet; for example, certain amounts of calcium and grit can reduce lead toxicity. Lead inhibits normal growth in birds, causes anemia, may interfere with normal sexual development, disrupts metabolism, and prevents the assimilation of nutrients.

Domestic and caged animals may suffer lead poisoning from atmospheric pollution in urban areas and from leaded paint. Lead poisoning particularly affects the central nervous and vasculatory systems of domestic mammals; gastric disorders are also common. Chronic exposure, through ingestion of six to seven milligrams of lead per kilogram of body weight, can result in poisoning in cattle. "Fallout" from lead smelters often contaminates vegetation grazed by livestock, thus poisoning them.

Environmental transport and transfers have an important bearing on the ecological effects of toxic substances and the fact that such movements are relatively sluggish and inefficient for lead probably explains the rather minimal ecological effects of the very large quantities of lead which have been dispersed to the environment over the years.

Lead in soil is relatively immobile and can persist for a long period of time irrespective of the form in which it is added. Maximum concentrations are found at the surface, and decrease in depth. Lead appears to be tightly bound by most soils, and substantial amounts must accumulate before it represents a hazard to the growth of higher plants. Lead is absorbed from the soil by plants, but is not accumulated to any great extent. Only small amounts of lead are translocated to the above ground portion of plants, the major portion remains in the roots. Plant uptake varies among plant species, and is also affected by plant age, pH and composition, soil texture and climate. Lead levels in leaves are generally related to atmospheric conditions.

The atmosphere is the major source of lead associated with above ground vegetation. Particulates attach to the rough surface and waxy cuticles of leaves; they may also become embedded in surface cells. Some foliar absorption and translocation may occur. Airborne lead deposited on vegetation and other food sources is a source of lead poisoning in animals when accumulations are excessive; however lead poisoning of animals is a rare occurrence in present-day environments.

Birds in urban areas, and insects and small mammals along heavily travelled roads have elevated body burdens of lead. Sediments in water bodies are a sink for lead. The lead content of sediments may be several thousand times that of the water. Aquatic biota, both invertebrate and vertebrate have been shown to bioconcentrate lead to levels greater than in the water, and sometimes similar to those in the sediments. However, concentrations of lead tend to decrease with increasing trophic level within aquatic systems, and there is essentially no indication of biomagnification of lead as it transfers through the trophic levels of the food chain.

Lead has been characterized by The Council on Agricultural Science and Technology (1976) as a nonessential element that exhibits a low degree of potential toxicity to plants and a high degree of potential toxicity to animals.

In the following paragraphs the ecological effects and transfers of lead are briefly discussed. A more detailed treatment is to be found in the recent Review of the Environmental Effects of Pollutants: VII.Lead (Bell, et. al., 1978), one of a series of REEP documents issued by EPA.

6.2 EFFECTS ON TERRESTRIAL AND AQUATIC PROTISTA

There are few available data concerned with the effects of lead on terrestrial or aquatic protista (bacteria, protozoans, algae, fungi, and viruses). The major effects of lead that have been reported include inhibitions of enzymes, biosynthetic reactions, and growth. However, no references were found on the effects of lead on viruses. The effects of inorganic and organic lead on protistan cells, especially bacteria, have implications for cellular processes in human beings. Alteration of biosynthetic pathways through direct or indirect actions on enzymes and their activities is probably the most significant effect of lead on any living organism.

Of the various protists that have been studied, only the bacteria are known to be capable of transforming certain inorganic and organic lead compounds into a volatile and more toxic tetramethyllead (Section 7.6.4) without affecting cell viability.

Lead interference with protistan populations may cause a disruption of their important activities in the environment -- nutrient cycling, decomposition of organic matter, mobilization and biotransformation of metals and inorganic and organic compounds, etc. A good case in point is that the toxic effects of lead could conceivably result in the disturbance or destruction of the lowest level of the aquatic food chain (algae) which would, in turn, have a direct effect on productivity at other levels of the food chain.

6.2.1 Terrestrial Protista

It has been hypothesized that observed reductions in litter decomposition and nutrient cycling in soils may have resulted from the accumulation of heavy metals in the litter and upper soil, hypothesized to result from interference with both enzymatic and direct microbial activity (Smith, 1976; Watson, 1975; Watson, et al., 1976.)

Cole (1977) conducted a series of experiments to determine if lead has any effect on soil microbes and if soil microorganisms alter the availability of lead to biological systems. He examined the effect of lead on enzyme synthesis when the appropriate substrate is added to soil. Amylase was used as the indicator enzyme because a large number of bacteria in cultivated soils produced this enzyme. The addition of 2 mg Pb^{2+}/g soil coincident with or after amendment with starch or maltose resulted in 75 and 50 percent decreases in net synthesis of amylase and α-glucosidase, respectively. Invertase synthesis in sucrose-amended soil was reduced after lead, as lead acetate (1.5 mg Pb^{2+}/g soil) was added. Two milligrams of Pb^{2+}/g of soil were sufficient to inhibit enzyme synthesis but not enzyme activity. The decrease in amylase synthesis was paralleled by a decrease in the number of lead-sensitive, amylase-producing bacteria but not in the soil fungi population.

The degree of inhibition of enzyme synthesis was related to the quantity and the specific form of lead added but not to the water solubility of the lead compound added. Lead oxide did not inhibit amylase synthesis at concentrations of 13 mg Pb^{2+}/g of soil whereas lead sulfate at 10.2 mg Pb^{2+}/g of soil reduced amylase synthesis to 35 percent of control levels after 48 hours. Lead acetate, lead chloride, and lead sulfide reduced amylase synthesis at total lead concentrations of 0.45 mg/g of soil or higher. The results indicated that lead is a potent but somewhat selective inhibitor of enzyme synthesis in soil, and that highly insoluble lead compounds (e.g., PbS) may be potent modifiers of soil biological activity.

Tyler, et al., (1974) found that 500 to 1000 micrograms of lead acetate per gram of meadow soil (clayish mull soil) significantly decreased nitrate levels but had no effect upon ammonium levels after 2 to 8 weeks of incubation. Since no supplemental nitrogen was added, the decrease in nitrate may have resulted from the inhibition of soil organic matter decomposition combined with decreased activity of soil-nitrifying bacteria.

When Bhuiya and Cornfield (1973) treated a sandy soil of pH 6.0 with 1000 ppm lead as lead oxide, carbon dioxide release was decreased to a moderate extent. The toxic effects of high lead levels on the activity of heterotrophic organisms attacking organic matter were probably due to the ability of lead to compete with essential elements (Mn, Fe, Mg) for active enzyme sites (-SH, $-NH_2$, =NH). Such competitive inhibition would account for the decrease in carbon mineralization of native soil organic matter.

There is some indication that lead has some inhibitory effects on a biochemical parameter (yet to be defined) in the biosynthesis of lipids in bacteria. The lipid composition of *Micrococcus luteus* cells was reduced by as much as 50 percent when cultured five or six times in culture media containing lead bromide or lead nitrate (Peterson, et al., 1975).

M. luteus became osmotically sensitive and lost carotenoids when exposed to lead bromide or lead chloride for 20 consecutive days (Tornabene and Edwards, 1973). In contrast, Tornabene and Edwards (1972) reported that lead bromide, lead iodide, and lead bromochloride (the principal component of automobile exhausts) in concentrations from 0 to 0.7 mg/ml had no detectable effects on the overall growth rate and cell viability of *Azotobacter* sp. and *Micrococcus luteus*.

Free lead ions (269 mg/l) slightly reduced biomass accumulation by the fungus *Aspergillus niger*. The chelates lead thiopicolinanilide and lead diethyldithiocarbamate have strong fungicidal properties as do tributyllead 2-ethyl hexanoate, tributyllead acetate, and triphenyllead acetate. (Zlochevskaya and Rukhadze, 1968)

6.2.2 Aquatic Protista

Some cells of *Aeromonas* sp., incubated for two weeks in nutrient media containing tetramethyllead acetate, became osmotically sensitive and leaked intracytoplasmic materials. This bacterium, however, has been shown to be capable of methylating trimethyllead acetate to the volatile tetramethyllead. It does have the ability to accommodate against toxic concentrations of organic lead salts by binding the lead to the cell envelope. However, some cells have been found to be sensitive to lead which suggests that they have lost this ability to exclude lead from the cell (Silverberg, et al., 1976).

Lead as 10^{-4}M lead acetate has been found to inhibit the incorporation of ^{14}C-leucine into *Escherichia coli*. It has been suggested that lead probably affects protein synthesis by its action on the synthesizing enzymes and by the hydrolysis of t-RNA and, supposedly, similar RNA species (Ulmer and Vallee, 1969). A glutaminase enzyme isolated from *E. coli* was inhibited by one millimole of lead acetate (Hartman, 1968).

Few studies have been conducted on the effects of lead on algae. The growth of the freshwater green alga *Chlorella* was inhibited by a lead concentration of 0.1 ppm. In contrast, the same lead concentration had no effect on the marine phytoplankters *Phaeodactylum tricornutum*, *Chaetoceros galvestonensis*, and *Cyclotella nana* (Rice, et al., 1973; Hannan and Patouillet, 1972). Cell numbers of *Chlorella pyrenoidosa* decreased when the alga was cultured in demineralized water containing 1.0 ppm lead as lead acetate. Ten ppm of lead (as lead acetate) in modified Bristol's solution reduced the number of algal cells by 52 percent after 5 days. (Banks, 1972).

Monahan (1973) found that lead (as lead chloride) concentrations of 50 to 500 ppm reduced cell density in cultures of the green alga *Hormotila blennista* in both light and dark environments. The median effective dose

(ED_{50}) of lead (as lead nitrate) which caused a 50 percent reduction of $^{14}CO_2$ fixation was 15 to 18 ppm for Anabaena sp. (blue-green alga), Chlamydomonas reinhardti (green alga), and Navicula pelliculosa (diatom), and 5 ppm for Cosmarium botrytis (desmid) (Malanchuk and Gruendling, 1973).

Lead (as lead chloride) concentrations of 2.5 to 10 ppm were sublethal to the marine green alga Platymonas subcordiformis, delayed cell division and daughter cell separation; however, a concentration of 60 ppm was lethal (Hessler, 1974; Hessler, 1975). Loss and alteration of flagella and impairment of motility also resulted from lead treatment.

Bonotto and Kirchmann (1973) noted that cap formation, which is necessary for reproduction in the marine alga Acetabularia mediterranea, in nucleate and anucleate cells was inhibited by lead nitrate (1 to 10 milligrams per liter) in four days.

Wong, et al. (1975) found that a momentary bubbling of less than 0.5 mg of lead as tetramethyllead (TML) into a culture of the green alga Scenedesmus quadricauda decreased primary productivity and cell growth by 85 and 32 percent, respectively. Cell exposed to TML tended to clump; similar results were obtained with another green alga Ankistrodesmus falcatus.

Lead, as lead acetate and lead nitrate, at concentrations of 42 to 500 ppm, has been found to kill and inhibit the growth of protozoan species such as Paramecium caudatum, Tetrahymena pyriformis, Chilomonas, and Blepharisma (Apostol, 1973).

6.3 EFFECTS ON PLANTS

Lead is not considered to be an essential element for plant growth and development and its effects on plants are rather uniform. Lead inhibits growth, reduces photosynthesis, interferes with cell division, inhibits enzymes unique to photosynthesis and respiration, reduces water absorption and transpiration, accelerates abscission or defoliation and pigmentation, and reduces chlorophyll and ATP synthesis.

Sensitivity to lead varies among plant species. The genetic constitution of the plant species, age of the plant, nutrient (e.g., phosphate) availability, soil pH, and cation exchange capacity of soils appear to be some of the responsible factors. In some cases, lead interacts synergistically with other toxic metal ions to induce toxicity.

Some of the lower plants (lichens, liverworts, and mosses) seem to have a pronounced, genetically-determined tolerance to lead. These plants are able to concentrate high lead levels as compared to environmental levels without any effects. In fact, mosses and lichens have been used to monitor atmospheric lead fallout and as a index for regional lead pollution (Ratcliffe, 1975; Skaar, et al., 1973; Höll and Hampp, 1975; Briggs, 1972). Spanish moss (Tillandsia usneoides), a vascular plant, has been used in the same capacity for the same reasons (Martinez, et al., 1971).

6.3.1. Metabolism, Uptake, and Absorption

Several investigators have studied the effects of inorganic lead on photosynthesis, respiration, transpiration and water uptake in plants. Net photosynthesis in detached sunflower leaves (*Helianthus annus*) was reduced by 50 percent when leaf tissue lead concentration was 0.93 micromole per gram (193 ppm). Transpiration was similarly reduced. The effects occurred primarily by lead interfering with the functioning of the stomata, the opening of which decreased log-linearly with increasing lead concentrations of 10 to 1000 micromoles (Bazzaz, et al., 1974a). Bazzaz, et al., (1974b) grew corn (*Zea mays*) and soybean (*Glycine max*) plants in media receiving lead chloride treatments which supplied 0 to 250 milligrams of lead per plant. At the 250-milligram level, photosynthesis was only 10 percent of maximum in soybeans, but was 47 percent in corn although the tissue lead content was much higher than than of soybeans. Transpiration exhibited trends similar to those of photosynthesis.

Höll and Hampp (1975) reported that 25 micromoles of lead applied to spinach leaves induced a pronounced inhibition of gas turnover with a greater inhibition of CO_2 uptake in the light period than of CO_2 release in the dark period. The intensity of $^{14}CO_2$ fixation by isolated spinach chloroplasts and ATP synthesis were reduced by lead concentrations of 2 to 200 micromoles.

The influence of lead ions on partial reactions of photosynthesis was investigated in some detail by Miles, et. al, (1972). They found that lead salts (chloride) inhibited electron transport in Photosystem II of the chloroplasts in spinach (*Spinacia oleracea*) and tomato (*Lycopersicum esculentum*) leaves but not in Photosystem I. Photosystem II is the site of oxidative reactions, and inhibition most likely occurs between the site of the primary electron donor and water oxidation. Bazzaz and Govindjee (1974) found that lead chloride may stimulate or inhibit Photosystem II activity depending upon the pH. At pH 7.8, 0.75 to 9 millimoles of lead chloride inhibited activity 16 to 39 percent; a stimulatory effect of 20 percent was observed with 9 millimoles of lead chloride at pH 6.5.

The action of lead on enzymes associated with the photosynthetic process can provide greater insight into the mechanism involved. In spinach root and leaf homogenates, several enzymes unique to the photosynthetic process exhibited changes in activities in the presence of 5 to 200 micromoles of lead nitrate. Five micromoles of lead nitrate inhibited ribulose diphosphate carboxylase, and lactate dehydrogenase showed a similar but less pronounced decrease in activity. In contrast, pyruvate kinase activity was enhanced by all lead nitrate concentrations and exhibited optimum activity at 20 micromoles. The NAD- and NADP- dependent triose phosphate dehydrogenase systems were mostly activated at the respective lead concentrations of 100 to 500 micromoles and 100 to 1000 micromoles. Concentrations exceeding those given above resulted in pronounced inhibition. Exposure of spinach leaves to less than 100 micromoles Pb(II) resulted in a pronounced inhibition of transpiration and water uptake (Höll and Hampp, 1975).

Koeppe and Miller (1970) and Miller and Koeppe (1971) found that lead affects mitochondrial respiration in corn. Lead chloride (50 to 62 millimoles per liter) stimulated the oxidation of exogenous reduced NADH by 174 to 640 percent whereas 12.5 millimoles per liter of lead chloride inhibited succinate oxidation by more than 80 percent. When inorganic phosphate was included in the reaction media, the subsequent addition of lead was not effective due to the low solubility of lead phosphate. When the addition of lead was followed by the addition of phosphate, succinate oxidation was no longer inhibited, however, the oxidation of exogenous reduced NADH continued to be stimulated (Koeppe and Miller, 1970).

The sensitivity to lead of cultured grasses varies greatly. Maize (Zea mays) and rye (Secale cereale) exhibited a lack of sensitivity to lead acetate concentrations of 10^{-10} to 100 milligrams of lead per liter. Barley, oats, and especially wheat showed symptoms of damage at even lower lead concentrations. Damage to barley (Hordeum vulgare) was visible, and death occurred four days after exposure to 0.1 M lead nitrate (Höll and Hampp, 1975). As little as 24 ppm of lead as lead chloride in sand culture stunted the growth of corn plants under phosphate-deficient conditions (Miller and Koeppe, 1971).

Bean (Phaseolus vulgaris) plants were visibly damaged 30 minutes after exposure to 0.1 M lead nitrate. There was a loss of turgor pressure and death after 24 hours. Phaseolus was damaged by 30 ppm lead as the sulfate alone, or in combination with other nutrient salts in solution culture (Höll and Hampp, 1975).

Radicle elongation of soil-grown maize seedlings was depressed by concentrations of 250 micrograms of lead per gram of soil (ppm) or 25 micrograms of cadmium per gram of soil when added singly. When added in combination, the magnitude of inhibition was greater than the sum of inhibitions when added separately. This phenomenon suggests synergistic interaction. The reason for the apparent interaction on maize root elongation was due, in part, to elevated metal concentrations which were found in the roots at higher treatment combinations. It is also possible that the selectivity of the cell membranes was reduced thereby allowing the rapid entry of metals (Hassett, et al., 1976). Miller, et al., (1975) found that lead and cadmium inhibited corn root elongation together, but not individually. The greatest reduction of growth occurred in soils with low cation exchange capacities and low pH. According to Bittell, et al., (1974), lead and cadmium do affect mitochondrial membrane permeability. Therefore, these metals may very well synergistically disturb the metobolic mechanisms unique to mitochondria which promote growth.

The root systems of loblolly pine (Pinus taeda) and red maple (Acer rubrum) seedlings became reduced and blackened while growing in two forest soils treated with lead chloride concentrations of 0.0002 to 0.02 M in water solutions. Accompanying those toxic symptoms were reductions in height, stem and root dry weights and root/shoot ratios, and increases in red pigmentation (anthocyanin content) and in casting of red maple leaves (Davis and Barnes, 1973).

The influence of lead on the root system is very pronounced. Root growth among sheep fescue (Festuca ovina) was retarded, markedly reduced, and stopped by 10, 30 and 100 ppm of lead nitrate, respectively. On the other hand, sheep fescue growing in a Knopp's culture medium showed little reduction in root growth at 25 ppm lead. It is believed that certain nutrients in the culture medium had an antagonistic effect on the toxicity of lead (Wilkins, 1957). A 93 percent inhibition of root ontogeny of germinating lettuce seeds was observed at a lead concentration of 0.001 M (Höll and Hampp, 1975). This probably is due to lead reducing the rate of mitosis in the root meristem cells. Perennial ryegrass (Lolium perenne var. S23) roots showed pronounced blackening at the tips (Jones, et al., 1973a).

Barker (1972) grew explanted tissues of cauliflower, lettuce, potato and carrot on media which contained varying concentrations of lead acetate. Lettuce growth was inhibited at 0.5 milligram per liter. The growth of carrot explants was significantly reduced at 0.005 milligram per liter and drastically inhibited at 5 milligrams per liter. Growth of cauliflower and potato was inhibited at lead levels between 0.5 and 5 milligrams per liter.

Mitotic inhibition accompanied by growth reduction was observed in onion (Allium cepa) and maize (Zea mays) when nutrition solutes contained increasing quantities of lead (Höll and Hampp, 1975). Radu (1971) found that at least 6 micrograms of lead ions per milliliter is a mitotic poison. In vivo experiments in which the onion (Allium cepa) and vetch (Vicia faba) were exposed to lead nitrate revealed that inorganic lead, like organolead, alters mitotic figures. Stathmokinetic effects were elicited. Lead concentrates higher than 6 micrograms per milliliter induced chromosomal modifications.

6.3.2. Yields

There are conflicting reports in the open literature as to the effects of lead on plant growth or yield. Höll and Hampp (1975) reviewed reports in which lead had been found to have increased the yield of barley, corn, wheat, buckwheat, and other crop plants. Sugar yield from sugar beets was increased by lead while the tuber mass and starch content of potatoes decreased. Lead nitrate was used in the above instances and it has been theorized that the positive effect on plant growth was entirely due to the nitrate ion.

When Garber (1974) conducted pot tests with soil containing 2,000 or 10,000 ppm lead plant growth and yield were reduced. Bush bean plants exposed to the highest soil lead concentration of 10,000 ppm had leaves which were only half as well developed as control plants and had reddish-brown mottling along the leaf veins. Newly-formed leaves were highly chlorotic. No root nodules developed on those roots exposed to 2000 ppm. Yields of bean pods were 75 and 7 percent of the control, respectively. A lead application of 1000 ppm singly as lead chloride, lead carbonate or lead nitriate or in combination significantly reduced lettuce yield whereas yields of oat top growth and roots were not affected (John and Van Laerhoven, 1972). Liming reduced the uptake of lead by the aboveground portions of lettuce and oats to a much greater extent than the uptake by oat roots.

Höll and Hampp (1975) and Zimdahl and Arvik (1973) cite research in which lead (as nitrate and acetate) in concentrations exceeding 0.01 percent reduced the germination of cress and mustard seeds; concentrations of 0.1 to 0.2 percent lead completely inhibited seed germination.

6.3.3. Organolead Compounds

Few data are available on the effects of organolead compounds. Of particular interest are those compounds which are used as antiknock additives in automotive gasoline. Siegel, et al., (1973) report that five Bougainvillaea sp. plants exposed to about 7 ppm tetraethyllead vapor at 25 C for a three-day period exhibited 14 percent defoliation or abscission. Fourteen days were required for total recovery from the three-day, organolead exposure. When Ahlberg, et al., (1972) exposed a clone of onion bulbs to varying concentrations of alkyl lead compounds, they noted disturbances of the mitotic spindle fiber mechanism at very low concentrations (10^{-6} and 10^{-7}M). The methyl compounds affected the spindle fiber mechanism to a much lesser extent than did the ethyl compounds.

6.4 EFFECTS ON AQUATIC BIOTA

6.4.1. Invertebrates

The effects of lead on invertebrates are very significant because they serve as an important food source for a large number of organisms which occupy higher trophic levels. The effects of lead vary from growth inhibition to death. Toxicity bioassays utilizing inorganic lead concentrations above the primary drinking water standard (50 ppb) reveal a wide range of LC_{50} values which, in many cases, depend on factors such as pH and hardness of water.

Lead nitrate (1 to 10 ppm) in natural seawater induced morphological changes and tissue reorganization of hydranths of the colonial coelenterate Eirene viridula (Karbe, 1972). Lead chloride impairs reproduction and is lethal to the microcrustacean Daphnia magna at levels ranging from 0.1 to 10 ppm (Biesinger and Christensen, 1975; McKee and Wolf, 1963). Lobsters kept in lead-lined tanks for 20 days died; those kept in steel-lined tanks survived 60 or more days (Wilder, 1952).

Environmental lead levels of 0.1 and 0.2 ppm were very toxic to Eastern and Pacific oysters and produced the following anatomical changes:

(1) Atrophy and diffusion of gonadal tissue;
(2) Less identifiable and lighter-colored hepatopancreases;
(3) Considerable edema; and
(4) Disappearing or less distinct mantle edges (Pringle, et al., 1968).

Calabrese et al., (1973) found that hard-shell clam embryos (Mercenaria mercenaria) are more sensitive to lead than are American or Eastern oyster embryos (Crassostrea virginica). For hard-shell clam embryos, the LC_0 for lead nitrate was 0.010 ppm as compared to 0.003 ppm for oyster embryos, the

LC_{50} values were 0.78 and 2.45 ppm for clam and oyster embryos, respectively. (Calabrese and Nelson, 1974). The significance of any level of toxicity among these economically important invertebrates is that the success of their populations depends upon the success of the embryos in the environment.

6.4.2. Fishes

Elevated concentrations (0.1-63 ppm) of lead and lead compounds have been found to be lethal or toxic to fishes (Table 6.1). Factors such as dissolved oxygen concentration, pH, duration of exposure, water temperature, and water hardness appear to be controlling factors which influence the toxicity of lead to fishes through changes in the rates of biological and chemical reactions.

Embryo and fingerling stages of fish are the most susceptible to lead toxicity. Highest mortality rates occurred among brook trout alevins (Salvelinus fontinalis) upon exposure to lead nitrate. Weights of the alevins decreased in response to exposures to lead concentrations of 134 and 525 ppb (Christensen, 1975). Embryos in the eggs of brook trout became severely deformed upon exposure to 500 ppb of lead. Similar deformations as well as an increased incidence of the blacktail syndrome were noted to have occurred in fathead minnows at 240 ppb (McIntyre, 1973). The growth of fingerling brook trout was reduced at lead concentrations of 25 mg/l/day administered as a slug dose under diel fluctuations of temperatures and dissolved oxygen concentrations (Dorfman and Whitworth, 1969).

Subtle effects result when fish are exposed to lead for extended periods of time. Dawson (1935) exposed catfish (Ameriurus nebulosus = Norturus nebulosus) to 5 ppm lead acetate (renewed every 48 hours) for 16 to 183 days. After several weeks, Dawson noted injured erythrocytes, progressive anemia, and a marked regenerative response in peripheral blood. The erythropoietic activity of the spleen was greatly reduced while the organ became smaller and paler in color. Those fish killed by the lead had extremely dilated gall bladders.

Rainbow trout, chronically exposed to lead (as lead nitrate) concentrations ranging from 0.00 to a maximum of 3,240 μg/l in hard water for 19 months, developed black tails, eroded caudal fins, and spinal curvatures. Some mortality as well as abnormal behavior (no interest in food, loss of equilibrium, lying on the bottom of the experimental tank) occurred at the highest level (3,240 μg/l) in hard water. Black tails in rainbow trout developed at lead concentrations of 360 and 12 μg/l in hard and soft water, respectively. Additionally, black tails, spinal curvatures and/or eroded caudal fins developed in the F_1 generation from brood-stock rainbow trout which had also been exposed to lead. After 6 months exposure to lead concentrations ranging from 3.1 to 95.2 μg/l in soft water, black tail ranged from zero to 100 percent; spinal curvature and eroded caudal fins ranged from zero to 8 percent. From these data the maximum acceptable toxic concentration (MATC) appeared to lie between 6 and 12 μg/l. (Davies and Everhart, 1973).

Table 6.1 CONCENTRATIONS OF LEAD REPORTED TOXIC OR LETHAL TO FISH[a]

Concentration of Lead, ppm	Type of Water	Time of Exposure	Species of Fish
0.1	--	--	Fish
0.1	--	--	Sticklebacks
0.1-0.2	Very soft	--	Sticklebacks
0.2	Soft	--	Fish
0.21	--	--	Guppy
0.25	Fresh	--	Fish
0.33	--	--	Minnows, brown trout and sticklebacks
0.34	1000-3000 mg/l of dissolved solids	48-hr TLm	Sticklebacks and coho salmon
1.0	--	Long term	Carp
1.0	--	8.5 days	Sticklebacks
1.4	Soft	18-24 hr	Rainbow trout
1.4	Tap	48-hr TLm	Bluegill sunfish
2.8	Fresh	--	Fish
4.0	Soft	10-12 hr	Rainbow trout
5.5	Stream water	--	Trout
6.3	Tap	24 & 28 hr TLm	Bluegill sunfish
10.0	--	--	Goldfish
17.0	--	--	Goldfish
27.0	Tap	16-183 days	Catfish
40.0	--	80-hr	Goldfish
63.0	--	80-hr	Goldfish

[a] Adapted from McKee and Wolf (1963)

The data presented show that acute or chronic exposures to lead represent a hazard in terms of interfering with fish growth and production. That lead interferes with embryonic development, the growth and development of young fish, and the behavior of fish is sufficient evidence to support the fact that lead in sufficient quantities (presumably levels greater than 50 ppb) will cause a decreased population success for one of man's most important food sources.

6.5 EFFECTS ON TERRESTRIAL BIOTA

6.5.1. Invertebrates

Decreased allele frequencies occurred at four isozyme loci in the vinegar fly, *Drosophila melanogaster*, at lead concentrations of 12 to 1200 ppm. The frequency changes were greatest in the first 5 kilometers from lead smelting facilities in Bixby, Missouri (Lower, 1975). These findings confirm the interference of lead with mitotic and meiotic processes.

6.5.2 Vertebrates

6.5.2.1 Birds --

The exposure of waterfowl to lead has been studied often with emphasis on the ingestion of lead shot. The use of shotguns in marshes and lakes by hunters results in the concentration of lead shot on the bottom of heavily hunted areas. The spent shot is eaten as is the available food. Stickel (1969) estimates that 2 to 3 percent of American waterfowl population dies annually from lead poisoning. Mortalities among Canadian and U.S. waterfowl (largely mallard, mottled, redhead, and black ducks) have been estimated by the U. S. Department of the Interior (1976) to be 1.6 to 2.4 million annually out of a fall migration population of 100 million ducks.

Lead poisoning among waterfowl is characterized by typical signs and lesions, often resulting in death. Fairly well advanced lead intoxication in waterfowl has the following signs:

(1) Varying degree of emaciation (loss of up to 40 percent of original body weight, and prominent keel portion of the sternum)
(2) Reduced activity with reluctance to fly
(3) Lowered food intake
(4) Palsy (wing droop)
(5) Bile staining of vent area
(6) Tendency to seek isolation and cover, and
(7) Loss of ability to walk or stand (U.S. Department of Interior, 1976).

Internal morphological changes associated with lead-poisoning in waterfowl include:

(1) Lack of fat
(2) Atrophy of striated muscle, liver and kidneys
(3) Excess fluid in the pericardial sac
(4) Distended gall bladder
(5) Atrophied gizzard with grinding pads hardened and bilestained
(6) Proventriculus impacted with food and grit, and
(7) Anemia and paleness of the whole body (U.S. Department of Interior, 1976).

Mallard drakes were exposed to particulate lead in four concentrations (0, 17.8, 89.0, and 178 g/m^2) in simulated marsh areas over a 14-week period. At necropsy those ducks exposed to 178 g/m^2 were emaciated and had impacted proventriculi, green-stained and eroded gizzard linings, atrophied livers, distended gall bladders, hydropericardia, and acid-fast intranuclear inclusions in kidney tubules. Those in the 89 g/m^2 treatment group had all but two lesions--distended gall bladders and hydropericardia (Irwin and Karstad, 1972).

Karstad (1971) found myocardial infarctions and lesions associated with fibrinoid necrosis of the media of arterioles and small arteries in 75 percent of 67 waterfowl poisoned by ingesting lead shot. Infarcts were also noted in striated muscles and the smooth muscles of the gastrointestinal tract.

Secondary lead poisoning can occur among the predatory birds. The prairie falcon _Falco mexicanus_ was poisoned when fed a diet of mallard duck heads containing at least one shot and/or lead residues of 2.0 to 55.8 ppm (muscle) and 4.7 to 32.6 ppm (brain) (Benson, et al., 1974). The same occurred with an Andean condor which had been fed hunter-killed cottontail rabbits, squirrels, and groundhogs (Locke, et al., 1969).

Diet influences the deposition of lead in tissues and the degree of toxic effects. Longcore, et al., (1974) showed that the presence or absence of grit, and the type of grit affected the toxicity of ingested lead shot. In ducks fed crushed oyster shell grit (which contains calcium), a rapid rate of shot erosion was somewhat offset by the concomitant increased chance of voidance of the lead shot. The net result was less, but more rapid, mortality among ducks with grit than in ducks without grit. Anderson and Stewart (1969) found that trace metals are less likely to induce toxic conditions in ring-necked pheasants when calcium is abundant in the soil.

Lead acetate fed to young Japanese quail was toxic at the level of 500 and 1000 ppm. (Morgan, et al., 1975). This toxicity was evidenced by an inhibition of normal growth and anemia. Additional data suggested that lead interfered with normal sexual development in male quail. Dosing adult bobwhite quail cocks with ten or more lead shot caused a significant increase in mortality as compared to controls. More than 90 percent of those dosed with 30 shot per week died by the end of four weeks (Damron and Wilson, 1975).

Lead chloride was found to be highly toxic to chick embryos (_Gallus domesticus_), giving survival rates of 74 to 23 percent when used at 0.001 to 5 ppm. Lead-treated survivors displayed a high frequency of morphological abnormalities (Birge, et al., 1975).

The end result of the changes caused by lead poisoning is that a bird has little or no intake of nutrients and cannot retain or process the nutrients it does have. Protein metabolism and, thus, the mechanisms involved with the immune response are most likely interfered with. An example is the increased mortality in chicks administered bacterial endotoxins and sublethal doses of lead acetate. In summary, lead-poisoned birds are subject to both predation and disease (Stickel, 1969; U.S. Department of Interior, 1976).

6.5.2.2 Mammals --

Leopards, a variety of other cats, primates, and other animals housed at the Staten Island Zoo exhibited the following symptoms of lead poisoning: death, hair loss, loss of muscular coordination, and paralyzation (Bazell, 1971). Tissue analyses revealed extremely high levels of lead. The major source of lead was thought to be atmospheric pollution but leaded paint was not discounted. The studies of Zook, et al., (1972) indicate a cause-effect relationship between leaded paint and lead intoxication of caged, wild animals at the National Zoological Park in Washington, D. C. Evidence that would verify the effect of lead on population dynamics of wild mammals or on individual wild mammals is lacking because of the difficulty involved in assessing cause-effect mortality factors which would affect wild mammals in nature.

In domestic animals, lead poisoning appears to affect all major organs and organ systems, particularly the central nervous and vascular systems. Chronic exposures to 6 to 7 milligrams of lead per kilogram of body weight day can result in toxicosis in cattle. Acute, lethal, single exposures are 400 to 600 milligrams of lead per kilogram of body weight for adult cattle (Buck, 1975). From 0.2 to 0.4 grams of lead (as lead acetate, carbonate, or oxide) per kilogram of body weight was sufficient to cause death in young calves in a few days (Allcroft and Blaxter, 1950). Gross brain lesions consisting of cerebrocortical softening, yellow discoloration, and cavitation have been observed in cattle subjected for more than nine days to naturally-occurring lead poisoning. Cattle with acute poisoning exhibited microscopic lesions which varied from diffuse capillary activation to scattered focal areas of status spongiosus and necrosis of the cerebral cortex (Christian and Tryphonas, 1971). Cattle with subacute and chronic poisoning had microscopic lesions ranging from different degrees of status spongiosus, astrocytic swelling, and nerve cell degeneration to severe cavitation and vascular proliferation.

Chronic lead poisoning in horses that had fed on contaminated vegetation within the "fallout" area of a lead smelter was characterized by anorexia, loss of body weight, muscular weakness, anemia, laryngeal hemiplegia, elevated urine lead concentration following chelation therapy, and inhalation pneumonia preceeding death (Knight and Burau, 1973). Schmitt, et al., (1971) reported depleted adipose tissues with generalized emaciation, edematous synovial membranes, erosive and rough joint surfaces, and exostoses of the articulating bones, especially metacarpals, diffuse edema and necrosis of individual nerve cells, hepatic cell degeneration, a slight to moderate degree of nephrosis, and intranuclear inclusion bodies in the epithelial cells of collecting renal tubules and in some of the hepatic cells in six horses that had ingested up to 264 ppm lead in forage.

Lead poisoning in young dogs is characterized by gastrointestinal dysfunction (colic, vomiting, and diarrhea) and nervous disorders (convulsions, hysteria, nervousness, and behavorial changes). There is no severe anemia but their blood has numerous stippled and immature nucleated erythrocytes (Zook, et al., 1969).

Although cats are resistant to the absorption of lead in large quantities, they succumb to the repeated administration of small doses (Clarke, 1973). When poisoned, cats exhibit gastric (anorexia, vomiting) and nervous (epileptiform convulsions) symptoms. They became very thirsty, and partially constipated and paralyzed with hyperaesthesia of the skin. Degenerative changes may occur in the liver and kidneys; the intestines become contracted; and the tongue and throat turn blue.

6.6 ENVIRONMENTAL INTERACTIONS

As indicated in Section 5.2 and 5.3, lead is ubiquitously distributed throughout the environment as a natural component as well as a contaminant released from a variety of anthropogenic sources (mines, smelters, refineries, manufacturers, etc.).

Although the input of lead by the combustion of leaded fuels is considered to be the major source of lead to the environment, other man-made sources as listed above also contribute this toxic metal to the environment. The fallout of lead-containing dusts and its removal by rain or snow contribute lead to soil and water. The lead will then either be retained in soil and sediments and become partially available for uptake by biota and/or biotransformation by protists or leached into the groundwater. Lead in sediments will generally be able to reenter the environmental cycle, except that ocean sediments serve as an ultimate sink for waterborne lead. The cycling of lead through various pathways and compartments of the environment is illustrated in Figure 6.1.

6.6.1 Physical and Chemical Behavior

Only a small fraction of the lead emitted to the atmosphere from natural and anthropogenic sources is carried away by the wind. Lead aerosols suspended in the atmosphere eventually fall to the ground and are transferred to the soil, water, plant, or animal compartment of the environment by sedimentation, impaction, precipitation (rainfall, snow) or inhalation. The mean residence time of lead aerosols in the atmosphere, calculated at 7 to 30 days, reflects the efficiency with which aerosols are removed by precipitation (National Academy of Sciences, 1972). The mean residence time for natural lead-210 is estimated tc be 9.6 days ± 20 percent (Zimdahl and Arvik, 1973).

There are few experimental data on the environmental behavior of inorganic forms of lead emitted from lead emission sources other than internal combustion engines; however, in many cases, the chemical form lost is that which is being handled or prepared. From combustion operations, e.g., waste or sludge incinerators or coal combustion, an oxide is a likely form.

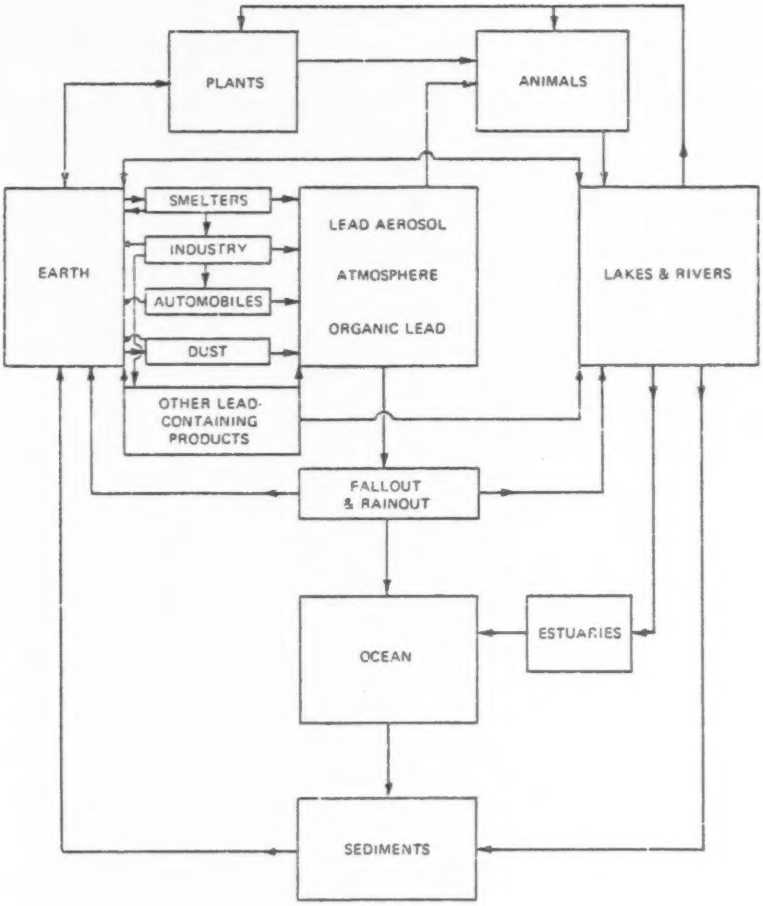


figure 6.1 Ecologic flow chart for lead showing possible cycling pathways and compartments

Source: National Academy of Sciences (1972).

The chemical form of lead in automobile exhausts changes rather rapidly as the components react with moisture, carbon dioxide, and sulfur dioxide in the atmosphere. PbBrCl is the principal initial component although there is evidence that exhaust particles are bromine-rich compared to stoichiometric PbBrCl (Boyer and Laitinen, 1975). Measurements made by Ter Haar and Bayard (1971) showed that the amount of $(PbO)_2$ PbBrCl in the exhaust is almost as great as that of PbBrCl. These two compounds accounted for 31.4 and 32.0 percent of the lead, respectively. Other halogen compounds and their percentages were: $PbCl_2$ (10.4), PbOHCl (7.7), $PbBr_2$ (5.5), $(PbO)_2$ $PbCl_2$ (5.2), PbOHBr (2.2), and $(PbO)_2$ $PbBr_2$ (1.1). The nonhalogen compounds found were: PbO_x(2.2), $PbCO_3$ (1.2), $(PbO)_2$ $PbCO_3$ (1.0), and $PbSO_4$ (0.1). After 18 hours, measurements showed that about 75 percent of the bromide and 30 to 40 percent of the chloride compounds had disappeared. The lead oxides, carbonate, and oxycarbonate then constituted 65 percent of the sample. On longer exposure to the atmosphere, the carbonate content increased and some of the lead was converted to sulfate. Particulate samples collected in the vicinity of a rural road which would represent "aged exhaust" had a lead distribution of $PbCO_3$ (30.2), $(PbO)_2 \cdot PbCO_3$ (27.5), PbO_x (20.5), $PbO \cdot PbSO_4$ (5.0), and $PbSO_4$ (3.2). The remaining 15 percent of the lead was distributed among the chloride and bromide compounds. Consequently, by the time lead aerosols are removed from the atmosphere, they are converted almost completely to carbonates and sulfates.

Lead in soil is relatively immobile and can persist for a long period of time whether added to the soil as halides, hydroxides, oxides, carbonates and sulfates from the combustion of automotive fuels or from industrial processes, as lead arsenate insecticides, or as an impurity in fertilizers and as a component of wastewaters (Tidball, 1976). The inorganic lead compounds from automobile exhausts are deposited within a few meters of the roadway and are restricted to the topsoil, presumably because the complex lead oxides and sulfates are or soon become relatively insoluble. As noted in Section 8.4.4, Olson and Skogerboe (1975) have shown that most of the exhaust particles in soil and road dust is lead sulfate, whose solubility is only 40 ppm (Table 3.3). Soils receive about 1 $\mu g/cm^2/yr$ from precipitation and 0.2 $\mu g/cm^2/yr$ may be deposited in dustfall (National Academy of Sciences, 1972). Although most deposition of lead on soil surfaces is derived from the combustion of leaded gasolines, even in remote non-industrialized areas, the concentration of lead near the surface is higher than in lower horizons. For example, Swaine and Mitchell (1960) studied the lead profiles in Scotland and found the lead content at 125 cm (50 in) to be one-half the surface value (this was before the large increase in gasoline anti-knock additives). Ter Haar (1975) postulates that plants take lead from the soil and this lead remains behind at the surface when the plant decays, and being held very tightly to the soil, does not leach into the lower horizons.

Hassett (1974) found that soil properties associated with increasing cation exchange capacity, i.e., higher organic matter content, higher surface area, and higher clay content, have a greater effect on lead sorption than soil pH, and that soil pH has a greater effect than soluble phosphorus. Cation exchange capacity was found to be the most significant factor affecting lead sorption from selected Illinois soils e.g., silty soam, silty clay loam, loamy sand.

The importance of indigenous soil phosphate as a potential buffer agent for lead in the soil solution has been suggested by the work of Nriagu (1972, 1973a, 1973b). His thermodynamic data suggest that the pyromorphite form of lead, i.e., $Pb_5(PO_4)_3X$, where X = halide ion, OH^-, etc., should be considered when studying the fate of lead. He concluded that in the presence of the chloride ion, $Pb_5(PO_4)_3Cl$ (chloropyromorphite) was the stable modification of lead in natural systems.

Nriagu (1972) reports that primary lead orthophosphate is a stable compound in highly acidic solutions. Its formation may, therefore, limit the stability field of chloropyromorphite in acidic environments. In studying the stability of the $PbCl_2$-PbO-P_2O_5-H_2O system under most natural environments, Nriagu (1973a) found that secondary lead orthophosphate ($PbHPO_4$) rapidly transforms into chloropyromorphite in the presence of chloride ions. Similarly, tertiary lead orthophosphate, $Pb_3(PO_4)_2$, transforms into chloropyromorphite. $PbHPO_4$ and $Pb_3(PO_4)_2$ are unstable relative to chloropyromorphite.

The most basic lead orthophosphate likely to be formed in alkaline systems is tetraplumbite phosphate, $Pb_4O(PO_4)_2$. It is also rapidly converted to chloropyromorphite in the presence of chloride ions. The limiting pH which induces the transformation varies from 11.6 to 13.6, the significance of which is doubtful because both $Pb_4O(PO_4)_2$ and $Pb_5(PO_4)_3Cl$ (chloropyromorphite) transform to lead oxides and hydroxides at pH values greater than 10.0. The fact that lead hydroxide is not commonly found with chloropyromorphite, however, suggests subsequent alteration of the $Pb(OH)_2$ to other phases (Nriagu, 1973a).

Santillan-Medrano and Jurinak (1975) conclude that in noncalcareous soils, the solubility of lead appeared to be regulated by $Pb(OH)_2$, $Pb_3(PO_4)_2$, $Pb_4O(PO_4)_2$, and $Pb_5(PO_4)_3OH$, depending on pH. In calcareous soils, $PbCO_3$, also assumed importance. $Pb(OH)_2$ appears to regulate Pb^{2+} activity when solution pH is less than about 6.6. As pH increases, formation of lead orthophosphate, lead hydroxypyromorphite and also tetraplumbite phosphate become a distinct possibility. In calcareous soils, general decreases in solubility occurred with increasing pH. In the pH range of 7.5 to 8.0, the possibility exists for the formation and/or mixture of $PbCO_3$, $Pb(OH)_2$, $Pb_3(PO_4)_2$, $Pb_5(PO_4)_3OH$ or $Pb_4O(PO_4)_2$.

The data of Santillan-Medrano and Jurinak (1975) support the contention of Nriagu (1972 and 1973a) that lead phosphate formation can serve as a sink for lead in ecosystems. This situation is particularly important where high levels of indigenous phosphate exist or in systems to which phosphate has been added.

In some special situations where initial soil lead concentrations are elevated, it apparently can be leached from the soil at a fairly rapid rate. Roberts and Goodman (1973) examined surface soils in the vicinity of smelters at intervals after permanent shutdowns of the smelters. Soil depletion of the lead was relatively fast. At two badly contaminated sites (104 and 328 μg/g of soil) the surface 5 centimeters lost lead at a rate that would reduce it to background values within 2 to 5 years.

Other evidence for fairly rapid removal of lead by leaching was obtained by Clark (1973) and an investigation of the leachate from landfills. Trace metals in the leachate were determined, and the lead concentration was found to be high as 500 ppb at a northern Illinois site. This value is ten times the interim primary drinking water standard and five times that permitted by the Illinois Polution Control Borad for domestic water supplies. It should be noted, however, that a landfill is not soil, and little opportunity exists for soil: lead interaction.

Lead has a marked propensity to form compounds of low solubility with the major anions of natural water. The hydroxide, carbonate, sulfide, and more rarely, the sulfate of lead may act as solubility controls (See Section 3.1). Hem and Durum (1973) compiled a list of the most stable dissolved forms of lead as calculated from thermodynamic data. These are Pb^{+2}, $PbOH^{+}$, $Pb(OH)_2$, $Pb(OH)_3^{-}$, $PbCl^{+}$, $PbCl_2$, PbO (crystal and crystalline phase), $Pb(OH)_2$ crystal, PbS crystal (cerussite), $Pb_3(OH)_2(CO_3)_2$ crystal (hydroxycarbonate or hydrocerussite) and $PbSO_4$ crystal.

There are few data on the interaction of lead with organic material in streams and lakes. Organic matter of stream sediments apparently binds lead and reduces its mobility as it does in soil, thus organic sediments will tend to decontaminate surface waters contaminated by lead.

Sediments in lakes, rivers, estuaries, and the ocean represent a sink for lead. If lead pollution of surface waters were to cease, the sediments would continue to supply lead to the water and its biota (Wershaw, 1976).

The pathways for the bioconversion or mobilization of lead from sediments are not well understood. Wong, et al., (1975) have shown, however, that microorganisms in lake sediments can transform certain organic and inorganic lead compounds into a volatile tetramethyllead. Although the conversion of lead nitrate and lead chloride (not lead hydroxide, lead cyanide, lead oxide, lead bromide, or lead palmitate) to tetramethyllead has been observed by Wong, et al., the conversion of inorganic lead to organic lead appears to be a rather difficult process, probably requiring specific physical, chemical, as well as biological conditions. Even direct chemical synthesis of methyllead compounds through alkylation of inorganic lead salts is very difficult because of the extreme instability of the postulated first intermediate monomethyllead salts. In contrast, the biotransformation of trimethyllead (as trimethyllead acetate) to tetramethyllead proceeds quite easily and has been demonstrated using microorganisms (Pseudomonas, Alicaligenes, Acinetobacter, Flavobacterium, and Aeromonas) isolated from the sediments of Lake Ontario. The bioconversion took place in lake water and nutrient media with or without sediment, and in the presence or absence of light. None of those microbes was able to produce tetramethyllead from inorganic lead in chemically defined media.

A possible chemical mechanism for the conversion of trimethyllead acetate to tetramethyllead in active, anaerobic sediment systems is the intermediate formation of trimethyllead sulfide followed by decomposition to give tetramethyllead (Jarvie, et al., 1975). Under anaerobic conditions, this mechanism appears to be dominant in the formation of tetraethyllead from triethyllead chloride. Jarvie, et al., believe that trialkyllead salts compete with inorganic sulfides for the necessary sulfur.

6.6.2 Environmental Transport and Transfers

All three media - air, water, soil - are involved in the environmental transport and inter-compartment transfers of lead introduced into the environment. It will facilitate the discussion to consider the media separately, first for plants and secondly, for animals, insofar as this is possible. Since many investigations examined more than one medium, there are overlaps.

6.6.2.1 Air/Plants--

Since the major source of lead to the biosphere is from the exhausts of automobiles, one area of concern is that of lead uptake by vegetation near well-traveled roadways and lead-producing or lead-utilizing industries. Lead content decreases with distance from these sources of lead and the concentration also decreases with depth in soil profiles. The data in Table 6.2 further summarize different findings of lead in the above-ground portion of grasses as related to distance from traffic or industry. Ter Haar (1970) investigated the effects of lead in air and soil on the lead concentrations of the washed edible and inedible portions of several important food crops. These were studied by growing crops in greenhouses supplied with filtered and unfiltered ambient air and in field plots planted in long rows perpendicular to a busy highway. On the basis of these crops (Tables 6.3 and 6.4), the conclusion was that lead occurring naturally in the soil is the main source of lead in the edible portions but that, in the concentration range studied, the variation of lead content of the soil did not affect the lead content of the crops.

Cannon (1976) cited a comprehensive study by the U. S. Environmental Protection Agency conducted in the Helena Valley area of Montana to determine the effects on vegetation of pollutants from a lead smelter. The lead content of the vegetation was found to decrease with distance from the stack. In one phase of the study, alfalfa, pinto beans, carrots, beets, petunias, and tobacco were grown in greenhouses in vermiculite, exposing the growing plants to ambient air, and in garden soils outdoors at the same distances from the stack. This was done in order to test how much lead might be absorbed from the soil and how much from the air in the vicinity of the smelter. The lead content of unwashed produce decreased with distance from the smelter. Assuming that the greenhouse-grown plants received the same atmospheric lead insult as did the plants grown outdoors, then it appears that half of the lead in the unwashed produce (3-6 ppm) came from the soil and half from the air.

Hemphill (1974) reports a comparison of lead accumulation by vegetable crops grown in the "new lead belt" counties in southeastern Missouri with those grown in north central Missouri areas with little auto traffic and no industrial activity. Lettuce, radishes, and green beans were planted. The

TABLE 6.2 LEAD IN THE ABOVE-GROUND PORTION OF GRASSES AS A FUNCTION OF DISTANCE FROM TRAFFIC OR INDUSTRIES

Distance from Road (m)	Lead Content (ppm, dry weight)	References
0 - 7.5	80[a]	Cannon and Bowles (1962)
7.5 - 15	66	
15 - 150	45	
Over 150	20	
0	664[b]	Motto et al. (1970)
7.5	454	
23	198	
37.5	139	
67.5	68	
Region of a smelter	500[c]	Rains (1971)
7.6 - 30	20 - 60	Chow (1970)
0 - 336.7	8 - 341 (fescue)	Hemphill et al. (1974)
0 - 336.7	5.8 - 344 (purple top)	
8 - 32 (U.S. 1, Beltsville, Maryland)	26.3 - 68.2	Lagerwerff and Specht (1970)
8 - 32 (Washington-Baltimore Parkway, Bladensburg, Maryland)	18.5 - 51.3	
8 - 32 (I-29, Platte City, Missouri)	7.5 - 21.3	
8 - 32 (Seymour Road, Cincinnati, Ohio)	7.6 - 31.3	

[a] Ash; average content.
[b] Highest values from unwashed grass samples.
[c] Highest value from a survey of four seasons.

TABLE 6.3 LEAD IN CROPS, GREENHOUSE STUDIES[a]

	μg/g dry weight[d]		
	Unfiltered Air	Filtered Air	LSD[b]
Air	1.45 $\mu g/m^3$	0.09 $\mu g/m^3$	–
Soil	17.1	17.1	–
Leaf lettuce	6.6[c]	3.2	1.3
Cabbage head	1.0	1.1	0.35
Cabbage–unharvested leaves	4.5	5.8	1.6
Tomatoes	0.59	0.72	0.26
Beans	1.4	1.2	0.52
Bean leaves	20.9[c]	7.9	8.8
Sweet corn			
Kernel	0.22	0.27	0.22
Cob	0.43[c]	0.69	0.24
Husk	6.9[c]	1.8	1.5
Carrots	1.7	2.1	1.9
Potatoes	0.30	0.33	0.12
Wheat	0.18	0.16	0.06

[a] Source: Ter Haar. Reprinted with permission from Environmental Science and Technology. (c) American Chemical Society, 1970.

[b] Least significant difference.

[c] Different from other values in row at 95 percent level of confidence.

[d] Washed crop

TABLE 6.4 LEAD IN CROPS, HIGHWAY STUDIES[a]

	μg/g dry weight[d]			
Feet from Road	30	120	520	LSD[b]
Air	2.3[c] $\mu g/m^3$	1.7 $\mu g/m^3$	1.1 $\mu g/m^3$	–
Soil	65[c]	40	25	–
Leaf lettuce	6.5	5.0	4.8	3.1
Cabbage head	0.56	0.86	0.83	0.44
Cabbage–unharvested leaves	6.4[c]	8.9	4.0	1.3
Tomatoes	1.3	1.2	1.6	1.3
Beans	1.9[c]	1.2	0.90	0.47
Potatoes	0.48	0.64	0.40	0.27
Sweet corn				
Kernel	0.39	0.21	0.83[c]	0.31
Cob	0.74	0.55	0.68	0.21
Husk	12.6[c]	6.6	5.7	3.6
Soybeans				
Beans	0.28[c]	0.12	0.10	0.10
Husk	15.9[c]	8.0	5.3	0.22
Oats				
Kernel	0.47	–	0.53	0.37
Chaff	31.4[c]	15.5	12.8	15.5
Carrots	1.6	–	1.5	0.61
Wheat				
Kernel	0.62	0.42	0.48	0.17
Chaff	17.8[c]	9.8	6.2	1.6

[a] Source: Ter Haar. Reprinted with permission from Environmental Science and Technology. (c) American Chemical Society, 1970.

[b] Least significant difference.

[c] Different from others at 95 percent level of confidence.

[d] Washed crop

average lead content (ppm dry weight) of the washed vegetables was as follows for the Lead Belt: lettuce leaves, 84-284; radish roots, 22-33; and green bean pods, 5.4-25. Lead Belt vegetable lead levels varied widely depending on proximity to mines and mills, smelters, and ore truck routes. In the control areas, the average lead content was: Lettuce, 20.6 ppm, radish root, 7.7 ppm; and green bean pods, less than 5 ppm.

The possibility of uptake into foliage cells from lead deposited on the leaves has not been separated from the possibility that particulates are only present as topical coatings which are resident on, embedded in, or fixed to the waxy cuticle of leaves. Retention may be a function of leaf morphology, pubescence, waxiness, and incident moisture but few experimental data are available. In addition, the role of the stomata is unclear, but it has been suggested that automobile exhaust particulates penetrate plants through the stomata (Zimdahl, 1976).

Using an aerosol generator and wind tunnel fumigation system, Wedding, et al., (1975) found that the gross deposition of lead aerosols on rough, pubescent leaves of sunflowers was ten times greater than on the smooth, waxy leaves of the tulip poplar. Page, et al., (1971) found that exposed tissues of plants with flexible, rough, hairy surfaces, and close to highways accumulated more lead per unit time than did those with smooth surface.

Heichel and Hankin (1972) found that lead, chlorine, and bromine associated with 7-micrometer particles are held on the rough surface or embedded in surface cells of the bark of trees. According to Heichel and Hankin (1976), rough white pine twigs retain lead particles more effectively than smooth needles. This characteristic has been observed with other coniferous species (Smith, 1971 and 1973). Heichel and Hankin (1976) found that roadside coniferous windbreaks function as sinks for vehicular lead emissions, thereby decreasing their dispersion from roads. The lead burden of older needles and twigs from white pine trees growing as a dense roadside screen west of a heavily and speedily traveled two-lane highway was consistently greater than that of younger twigs and needles.

Living plants were challenged by aerosols of lead bromochloride in an exposure chamber. Virtually all of the lead added to plants in this manner was removed by chloroform rinses or mild acid washes, suggesting that lead remained as a topical coating on the foliage. No significant amounts could be found in roots, fruits, or new shoot growth following exposure to lead aerosols. (Arvik and Zimdahl, 1974a).

Rabinowitz (1972) reported foliar absorption and translocation of lead halide aerosols by oats and lettuce grown near a freeway. Four to 10 ppm were absorbed by the leaves and translocated downward to the central taproots.

In contrast to vascular plants, mosses passively take up lead from precipitation and settled dust. Depending on the species of moss, lead ions have been found incorporated in the cell nucleus, cell wall, and organelles of nuclear and mitochondrial fractions or bound to cell-wall exchange sites without cytoplasmic penetration (Ratcliffe, 1975, Ruhling and Tyler, 1970; Skaar, et al., 1973; Brown and Bates, 1972; Roberts and Goodman, 1973.)

Although the occurrence of foliar uptake has been shown, the significance of the process has not been established. Nevertheless, foliar deposits from airborne lead cannot be ignored because if livestock graze on contaminated pastures or are fed lead-contaminated forage, or if man eats poorly washed vegetables and fruits, significant quantities of lead may be ingested.

6.6.2.2 Soil/Plants--

Plants take up lead in the ionic form from soils. Lead is absorbed, but not accumulated to any great extent, by plants from soil. Lead is either unavailable to plants or "fixed" in the roots with only small amounts being translocated to the above-ground portions even when plants are grown in soil containing substantial amounts of lead. This is due not only to the solubility of lead in the soil, but to some internal factors which govern the mobility of lead within the plant. Such a phenomenon varies among plant species. Variations in plant uptake of lead are due to plant age and species, soil phosphorus, organic matter content, pH, soil texture, climate, topography, pollution, and geological history of the soil. The amount of lead taken up from soil decreases as pH, cation-exchange capacity, and available phosphorus of the soil increase. (Council for Agricultural Science and Technology, 1976). Only about 0.003 to 0.005 percent of total soil lead is available for plant uptake (Zimdahl, 1976).

Jones, et al., (1973a) studied the uptake of lead from solution by ryegrass. They found that the roots of actively growing ryegrass provide a barrier which restricts the movement of lead to the above-ground parts of plants, and so to animals and man. Lead increases in ryegrass, cocksfoot, and mixed pasture grasses were confined to months of restricted growth. Lead was transported to the stems prior to winter dormancy possibly by way of the phloem (Mitchell and Reith, 1966). This same phenomenon was observed in leaves of deciduous trees and in wild oats (Rains, 1971; Zimdahl and Arvik, 1973).

Hampp and Höll (1974) verified an apoplastic lead transport from the roots of hardwoods with the transpiration stream. Young twigs from Acer platanoides trees growing in high density traffic areas showed that there is also phloem transport. Höll and Hamp (1975) indicate that since amino acids are the main component transported in the phloem, they possibly chelate lead ions, thereby making lead transport in the phloem a possibility even in the presence of high phosphate concentrations. Parenchymatous translocation can also occur (Hampp and Höll, 1974). Thirty percent of lead in the sapwood of black locust trees is thought to be in a mobile form and to be transported in a centrifugal direction during the growth of sapwood in each vegetation period. In nonheartwood-forming species (Tilia cordata and Acer platanoides), the uniform distribution of lead across the trunk by radial translocation was demonstrated in cross sections.

Several hydroponic experiments clearly indicate that lead quickly becomes associated with roots after treatment is initiated and that some of the lead is absorbed and translocated within the plant. However, the major portion remains in the roots (Zimdahl, 1976). Hydroponic studies have also shown that lead uptake is passive with (1) a rapid influx and attainment of a concentration plateau, (2) pH dependence, (3) relative insensitivity to metabolic inhibitors, and (4) no unusual temperature effects (Arvik and Zimdahl, 1974b).

Roots of hydroponically grown corn acquired a surface lead precipitate and slowly accumulated lead crystals in the cell walls (Malone, et al., 1974). Lead entering the roots was concentrated in some of the dictyosome vesicles which fused with one another to encase the lead deposit. The vesicles then moved to the periphery of the cell to fuse with the cell wall outside the plasmalemma. Similar deposits were observed in stems and leaves, suggesting that a similar process occurs in all plant tissues.

In experiments designed to determine the relative contribution of lead in soil, water, and air to that in perennial ryegrass and radishes, Dedolph, et al., (1970) found that ryegrass derived 2 to 3 ppm from soil sources. Leaf-lead levels in excess of this (measuring up to 15 ppm) were derived from, and quantitatively related to, atmospheric lead concentrations. Lead added to water at 40 micrograms per liter as $PbCl_2$ did not affect the concentration in the grass. Dedolph, et al., concluded that there are substantial amounts of soil-derived lead within plants and that soil has long been and remains an important source of lead in plants.

The extent to which lead moves within plants is questionable but deposition, translocation, and uptake studies make it clear that under certain conditions, lead is mobile within plants. Baumhardt and Welch (1972) found that lead in corn forage significantly increased with lead applications. Lead was absorbed by roots and translocated to plant tops but it was not translocated from stover to grain.

Data on lead accumulation and transport are supportive of the fact that lead is not generally translocated in large quantities to edible portions of plants. The problem remains, however of lead being deposited as a topical coating on vegetation.

At the present soil levels (due to pollution by tetraethyllead) and at levels likely to occur in the near future, lead does not appear to have an economic effect on crop growth or quality. (Zimdahl and Hassett, 1977).

6.6.2.3. Plants/Animals--

Evidence that lead can be and does move between terrestrial plants and terrestrial animals has been obtained by Price, et al., (1974). They found that in areas of high lead emissions from automobile exhausts, insect species that suck plant juices, chew plant parts, and prey on other insects had 10.3, 15.5, and 25.0 ppm lead, oven dry weight, respectively. Dorn, et al., (1974and 1975) found that cattle were poisoned after having fed upon vegetation contaminated with up to 100 ppm of lead.

Although there are no supportive data, lead burdens of upland game mammals (e.g., deer, rabbits, squirrels) and herbivorous small mammals are likely to be related to the lead content on or in the vegetation on which they feed. This is especially true in those areas affected by industrial and automotive fallout. Their mobility within given areas and/or ability to remove themselves from contaminated areas will determine, in part, the degree of exposure to lead by feeding. The movement of lead from terresterial plants to man is via consumption of contaminated fruits and vegetables.

Starlings sampled throughout the United States were found to have whole-body lead residues (wet-weight basis) of 0.4 ppm (Yakima, Washington) to 13.3 ppm (Chicago, Illinois) with a mean level of 3.18 ppm for all sample sites (Martin, 1972). Highest levels were found in starlings from urban areas, probably reflecting greater exposure to automotive and industrial contamination. Tansy and Roth (1970) found the same pattern of whole-body residues among pigeons from the City of Philadelphia. Blood-lead levels, however, of city pigeons were as low as, if not lower than, their country counterparts.

Small mammals which inhabit roadside areas are exposed to increased lead levels from automobile exhausts. In general, it has been found that lead levels in roadside mammal populations are higher than those levels found in populations some distance from roadways. Supportive data are summarized in Table 6.5. Jeffries and French (1972) found that although there was a 9.2 times difference in lead concentrations in the vegetation of major road verges and field areas (307 to 33 ppm, dry weight, respectively), there was only a 1.7 times (or lower) difference in the concentrations in mammals. Mammals living on the road verges had a mean lead level of 4.2 ppm, dry weight (1.3 ppm, wet weight).

Nymphs and adults of the European mantid (Mantis religiosa) were collected along I-83 (Baltimore, Maryland's major north-south freeway) which had an average daily traffic count of 13,000 motor vehicles in 1970 and in a control area 480 meters west of the freeway. In the control area, mantid nymphs had 2.0 ppm lead and breeding adults, 1.9 ppm. Nymphs in the freeway area had 7.35 ppm lead and the adults had 14.8 ppm (Giles, et al., 1973). A comparison of lead levels in the mantid nymphs and adults from the freeway and control areas reveals that measurable amounts of lead were being concentrated by these insect predators.

6.6.2.4 Aquatic Systems--

Freshwater vegetation and leaf litter material bind large quantities of lead. It is bound to anionic sites on the surface of aquatic plants or leaf litter by ion exchange (Gale, et al., 1973 and 1975). In spite of the large quantities of lead bound by vegetation and leaf litter in the streams of Missouri's New Lead Belt, biomagnification of lead was not found in the different aquatic organisms. Rather, elevated total body lead levels in fish, tadpoles, crayfish, and other aquatic arthropods were associated with gastrointestinal contents or with some additional lead bound or chelated to the mucous membranes of gills or to the mucous covering body surfaces.

The environmental fate and effects of cadmium and lead were studied in a laboratory model ecosystem with a terrestrial/aquatic interface, using silica sand, Bloomfield soil (sandy loam) and Drummer soil (silty clay loam) as substrates (Lu, et al., 1975). Applications were made directly to the substrates as lead and cadmium chloride and as sewage sludge as a source of heavy metals. The mobilization and incorporation of cadmium and lead into the food chain organisms were proportional to sorption capacity of the substrate and were highest in silica sand and lowest in Drummer soil.

Table 6.5 MEAN CONCENTRATION OF LEAD IN TISSUE OF SMALL MAMMALS FROM ROADSIDE AND CONTROL SITES
(ppm, dry weight)

Organism	Tissue	Control Site	Roadside Site	References
Deer mouse *Peromyscus maniculatus*	Brain	0.14	<0.57 - 0.84	Mierau & Favara (1975) and
	Lung		<0.58	Welch & Dick (1975)
	Liver	1.06	0.92 - 3.29	
	Stomach		1.64	
	Kidney	3.28	1.87 - 8.46	
	Muscle		0.49	
	Bone	4.84	14.0 - 52.1	
	Whole body	2.7		Schlesinger & Potter (1974)
White-footed mouse (*Peromyscus leucopus*)	Whole body	2.6	4.4	Quarles et al.,(1974)
Meadow vole (*Microtus pennsylvanicus*)	Whole body	4.9	8.6	Quarles et al.,(1974)
Short-tailed shrew (*Blarina brevicauda*)	Whole body	2.6 - 5.4	14.0	Quarles et al.,(1974); Schlesinger & Potter (1974)
Shrew (*Sorex* sp.)	Hair (unwashed)	6.0	235.6	Raymond & Forbes (1975)
Deer mouse (*Peromyscus maniculatus*)				
Townsend chipmunk (*Eutamias townsendii*)				
Meadow vole (*Microtus* sp.)				

Banus, et al., (1975) found that lead entering a salt-marsh ecosystem that had been treated with two levels (8.4 and 25.2 grams per square meter) of metal-containing sludge fertilizers was largely retained by the surface sediments. Six to 8 percent was taken up by marsh grasses. Lead in grass tops was exported as detritus to deeper waters but this loss was only about 3 to 4 percent of the entering lead. Even in a natural unpolluted marsh, the lead content of sediments was higher than that of the organisms living therein (Drifmeyer, 1975).

The common reed, salt-marsh cordgrass, and saltmeadow hay accumulate lead from salt marshes containing heavy-metal-containing dredge spoil (Drifmeyer and Odum 1975). Banus, et al., (1975) found that lead concentrations in shoots of cordgrass increased with increased salt-marsh contamination via human activity or experimental additions of fertilizer.

Drifmeyer (1975) and Drifmeyer and Odum (1975) have verified that lead from polluted, dredged material enters the food web in a dredge-spoil pond ecosystem and is transferred without biological magnification. Concentrations of lead tended to decrease markedly with increasing trophic levels in both detritus-based and grazing food chains.

It has been established that sediments in various bodies of water are a sink for lead (see Section 8.4.4). Absorption to sediments occurs as lead is presumably tied up by organic matter and hydrous metal oxides. However, as the lead content of the water decreases, the sediments contribute amounts to surface water so as to maintain an equilibrium. Mathis and Cummings (1971) reported that the lead content of sediments in the Illinois River was approximately 10,000 times greater than water. The lead content in clams, worms, and fish was much higher than that in water. Worms contained lead levels of the same magnitude as that found in the bottom sediments. The fish had considerably lower concentrations of lead, with the carnivorous fish having lower concentrations than noncarnivorous fish.

6.7 REFERENCES

Ahlberg, J., C. Ramel, and C. A. Wachtmeister. 1972. Organolead Compounds shown to be Genetically Active. AMBIO. 1(1):29-31.

Allcroft, R. and K. L. Blaxter. 1950. Lead as a Nutritional Hazard to Farm Livestock. V. The Toxicity of Lead to Cattle and Sheep and an Evaluation of the Lead Hazard under Farm Conditions. J. Comp. Pathol. and Therapeutics. 60:209-218.

Anderson, W. L. and P. L. Stewart. 1969. Relationships between Inorganic Ions and the Distribution of Pheasants in Illinois. Wildlife Management. 33(2):254-270.

Apostol, S. 1973. A Bioassay of Toxicity Using Protozoa in the Study of Aquatic Environment Pollution and Its Prevention. J. Environm. Res. 6(4):365-372.

Arvik, J. H. and R. L. Zimdahl. 1974a. Barriers to the Foliar Uptake of Lead. J. Environ. Qual. 3(4):369-373.

Arvik, J. H. and R. L. Zimdahl. 1974b. The Influence of Temperature, pH, and Metabolic Inhibitors on Uptake of Lead by Plant Roots. Environ. Qual. 3(4):374-376.

Banks, J. L. 1972. The Effect of Lead Acetate on Cells and Chlorophyll Content of Chlorella pyrenoidosa. M. S. Thesis, The Ohio State University, Columbus, Ohio. 56 pp.

Banus, M., I. Valiela, and J. M. Teal. 1974. Export of Lead from Salt Marshes. Marine Poll. Bull. 5(1):6-9.

Barker, W. G. 1972. Toxicity Levels of Mercury, Lead, Copper, and Zinc in Tissue Culture Systems of Cauliflower, Lettuce, Potato, and Carrot. Cana. J. of Bot. 50(5):973-976.

Baumhardt, G. R. and L. F. Welch. 1972. Lead Uptake and Corn Growth with Soil-Applied Lead. J. Environ. Qual. 1(1):92-94.

Bazzell, R. J. 1971. Lead Poisoning: Zoo Animals May be the First Victims. Science. 173(3992):130-131.

Bazzaz, M. B. and Govindjee. 1974. Effects of Lead Chloride on Chloroplast Reactions. Environ. Letters, 6(3):175-191.

Bazzaz, F. A., R. W. Carlson, and G. L. Rolfe. 1974a. The Effect of Heavy Metals on Plants: Part I. Inhibition of Gas Exchange in Sunflower by Pb, Cd, Ni and Tl. Environ. Poll. 7(4):241-246.

Bazzaz, F. A., G. L. Rolfe, and P. Windle. 1974b. Differing Sensitivity of Corn and Soybean Photosynthesis and Transpiration to Lead Contamination. J. Environ. Qual. 3(2):156-158.

Bell, M. A., R. A. Ewing, and G. A. Lutz. 1978. Review of the Environmental Effects of Pollutants: VII. Lead, EPA 600/1-78-029, U. S. Environmental Protection Agency, Health Effects Research Laboratory, Cincinnati, Ohio. Various Pagination.

Benson, M. W., B. Pharaoh, and P. Miller. 1974. Lead Poisoning in a Bird of Prey. Bull. Environ. Contam. Toxicol., 11(2):105-108.

Bhuiya, M. R. H. and A. H. Cornfield. 1973. Effects of Addition of 1000 ppm Cu, Ni, Pb and Zn on Carbon Dioxide Release during Incubation of Soil Alone and After Treatment with Straw. Environ. Poll. 3(3):173-177.

Biesinger, K. E. and G. M. Christensen. 1972. Effects of Various Metals on Survival, Growth, Reproduction, and Metabolism of Daphnia magna. J. Fisheries Res. Board Cana. 29(12):1691-1700.

Birge, W. J., A. G. Westermann, and O. W. Roberts. 1975. Lethal and Teratogenic Effects of Metallic Pollutants on Vertebrate Embryos. In: Trace Contaminants in the Environment, Proceedings of the 2nd Annual NSF-RANN Trace Contaminants Conference, Asilomar, Pacific Grove, California, August 29-31, 1974. T. Novakov (chairman), pp 316-320.

Bittell, J. E., D. E. Koeppe, and R. J. Miller. 1974. Sorption of Heavy Metal Cations by Corn Mitochondria and the Effects on Electron and Energy Transfer Reactions. Physiolo. Plantarum. 30:226-230.

Bonotto, S., and R. Kirchmann. 1973. Lead Effects on the Morphogenesis of the Unicellular Marine Alga Acetabularia. Annales de Gembloux. 79(2):121-130.

Boyer, K. W. and H. A. Laitinen. 1974. Lead Halide Aerosols, Some Properties of Environmental Significance, Environmental Science and Technology 8(13):1093-1096.

Briggs, D. 1972. Population Differentiation in Marchantia polymorpha L. in Various Lead Pollution Levels. Nature. 238(5360):166-167.

Brown, D. H., and J. W. Bates. 1972. Uptake of Lead by Two Populations of Grimmia doniana. J. Bryology. 7(2):187-193.

Buck, W. B. 1975. Toxic Materials and Neurologic Disease in Cattle. J. Amer. Vet. Med. Assoc. 166(3):1468-1472.

Calabrese, A., R. S. Collier, D. A. Nelson, and J. R. MacInnes. 1973. The Toxicity of Heavy Metals to Embryos of the American Osyter Crassostrea virginica, Marine Biol. 18(3):162-166.

Calabrese, A. and D. A. Nelson. 1974. Inhibition of Embryonic Development of the Hard Clam, Mercenaria mercenaria, by Heavy Metals, Bull. Environ. Contam. Toxicol. 11(1):92-97.

Cannon, H. L. 1976. Lead in Vegetation. In: Lead in the Environment. Geological Survey Professional Paper 957. T. G. Lovering (ed.) U. S. Government Printing Office, Washington, D. C., pp 53-72.

Cannon, H. L. and J. M. Bowles. 1962. Contamination of Vegetation by Tetraethyl Lead. Science. 137(3532):765-766.

Chow, T. J. 1970. Lead Accumulacion in Roadside Soil and Grass. Nature. 225(5229):295-296.

Christensen, G. M. 1975. Biochemical Effects of Methylmercuric Chloride, Cadmium Chloride and Lead Nitrate on Embryos and Alevins of the Brook Trout Salvelinus fontinalis. Toxicol. Appl. Pharmacol. 32(1) 225(5229):295-296.

Christian, R. G. and L. Tryphonas. 1971. Lead Poisoning in Cattle: Brain Lesions and Hematologic Changes. Amer. J. Vet. Res., 32(2):203-216.

Clark, T. P. 1973. Determination of Trace Element Levels in Landfill Leachate by Ion-Exchange, X-ray Spectrography. In: Trace Substance Environmental Health-VII, Proceedings of the 7th Annual Conference Trace Substances in Environmental Health, University of Missouri, Columbia, Missouri, June 12-14, 1973, D. D. Hemphill (ed.), pp 401-408.

Clarke, E. G. C. 1973. Lead Poisoning in Small Animals. Small Animal Practice. 14(4):183-193.

Cole, M. A. 1977. Lead Inhibition of Enzyme Synthesis in Soil. Appl. Environ. Microbiolo. 33(2):262-268.

Council for Agricultural Science and Technology. 1976. Application of Sewage Sludge to Cropland: Appraisal of Potential Hazards of The Heavy Metals to Plants and Animals. Report No. 64. Ames, Iowa. 63 pp.

Cox, W. J. and D. W. Rains. 1972. Effect of Lime on Lead Uptake by Five Plant Species. J. of Environ. Qual. 1(2):167-169.

Damron, B. L., and H. R. Wilson. 1975. Lead Toxicity of Bobwhite Quail. Bull. Environ. Contam. Toxicol. 14(4):489-496.

Davies, P. H. and W. H. Everhart. 1973. Effects of Chemical Variations in Aquatic Environments, Vol. III, Lead Toxicity to Rainbow Trout and Testing Application Factor Concept. EPA-R3-73-011c, U. S. Environmental Protection Agency, Office of Research and Monitoring, Washington, D. C., 80 pp.

Davis, J. B. and R. L. Barnes. 1973. Effects of Soil-Applied Fluoride and Lead on Growth of Loblolly Pine and Red Maple. Environ. Poll. 5(1):35-44.

Dawson, A. B. 1935. The Hemopoietic Response in the Catfish, Ameiurus nebulosus, to Chronic Lead Poisoning. Biol. Bull. 68(3):335-346.

Dedolph, R., G. Ter Haar, R. Holtzman, and H. Lucas, Jr. 1970. Sources of Lead in Perrenial Ryegrass and Radishes. Environ. Sci. Technol. 4(3):217-223

Dorfman, D. and W. R. Whitworth. 1969. Effects of Fluctuations of Lead, Temperature, and Dissolved Oxygen on the Growth of Brook Trout. J. Fish. Res. Board of Can. 26(9):2493-2501.

Dorn, C. R., P. E. Phillips, J. O. Pierce, II, and G. R. Chase. 1974. Cadmium, Copper, Lead and Zinc in Bovine Hair in the New Lead Belt of Missouri. Bull. of Environ. Contam. and Toxicol. 12(5):626-632.

Dorn, C. R., J. O. Pierce, G. R. Chase, and P. E. Phillips. 1975. Environmental Contamination by Lead, Cadmium, Zinc, and Copper in a New Lead-Producing Area. Environ. Res. 9(2):159-172.

Drifmeyer, J. E. 1975. Lead Enters Food Web of Estuarine Organisms. World Dredging and Marine Const. 11(12):41-43.

Drifmeyer, J. E. and W. E. Odum. 1975. Lead, Zinc, and Manganese, in Dredge-Spoil Pond Ecosystems. Environm. Conserv. 2(1):39-45.

Gale, N. L., P. Marcellus, and G. Underwood. 1975. Life, Liberty and the Pursuit of Lead: The Impact of Lead Mining and Milling Activities on Aquatic Organisms. In: Trace Contaminants in the Environment, Proceedings of the 2nd Annual NSF-RANN Trace Contaminants Conference Asilomar, Pacific Grove, California, August 29-31, 1974, T. Novakov (chairman), pp 295-307.

Gale, N. L., B. G. Wixson, M. G. Hardie, and J. C. Jennett. 1973. Aquatic Organisms and Heavy Metals in Missouri's New Lead Belt. Water Resources Bull., 9(4):673-688.

Garber, K. 1974. Heavy Metals in Air Pollution-Lead-Zinc-Cadmium-Influence on Vegetation. Staub Reinhaltung der Luft (in English), 34(1):1-8.

Giles, F. E., S. G. Middleton, and J. G. Grau. 1973. Evidence for the Accumulation of Atmospheric Lead by Insects in Areas of High Traffic Density. Entomol. 2(2):299-300.

Hampp, R. and W. Höll. 1974. Radial and Axial Gradients of Lead Concentration in Bark and Xylem of Hardwoods. Arch. Environ. Contam. Toxicol. 2(2):143-151.

Hannan, P. J. and C. Patouillet. 1972. Effect of Mercury on Algal Growth Rates. Biotechnology and Bioengineering. 14(1):93-101.

Hartman, S. C. 1968. Glutaminase of Escherichia coli. I. Purification and General Catalytic Properties. J. Biol. Chem. 243(5):853-863.

Hassett, J. J. 1974. Capacity of Selected Illinois Soils to Remove Lead from Aqueous Solution. Commun. Soil Sci. Plant Anal. 5(6):499-505.

Hassett, J. J., J. E. Miller, and D. E. Koeppe. 1976. Interaction of Lead and Cadmium on Maize Root Growth and Uptake of Lead and Cadmium by Roots. Environ. Poll. 11(4):297-302.

Heichel, G. H. and L. Hankin. 1972. Particles Containing Lead, Chlorine, and Bromine Detected on Trees with an Electron Microprobe. Environ. Sci. Technol. 6(13):1121-1122.

Heichel, G. H. and L. Hankin. 1976. Roadside Coniferous Windbreaks as Sinks for Vehicular Lead Emissions. J. Air Poll Control Assoc. 26(8): 767-770.

Hem. J. D. and W. H. Durum. 1973. Solubility and Occurrence of Lead in Surface Water. J. Amer. Water Works Assoc. 65(8):562-568.

Hemphill, D. D. 1974. Accumulation of Toxic Heavy Metals by Plants in Missouri's New Lead Belt. In: Trace Element Metabolism in Animals-2, W. G. Hoekstra, J. W. Suttie, H. E. Ganther, and W. Mertz, (eds.), University Park Press, Baltimore, Maryland, pp. 458-460.

Hemphill, D. D., C. J. Marienfield, R. S. Reddy, and J. O. Pierce. 1974. Roadside Lead Contamination in the Missouri Lead Belt. Arch. Environ. Health. 28(4):190-194.

Hessler, A. 1974. The Effects of Lead on Algae. I. Effects of Pb on Viability and Motility of 'Platymonas subcordiformis' (Chlorophyta: Volvocales). Water, Air, and Soil Poll. 3(3):371-385.

Hessler, A. 1975. The Effects of Lead on Algae. II. Mutagenesis Experiments on Platymonas subcordiformis (Chlorophyta: Volvocales). Mutation Research, 31(1):43-47.

Höll, W. and R. Hampp. 1975. Lead and Plants. Residue Reviews. 54:79-111.

Irwin, J. C. and L. H. Karstad. 1972. The Toxicity for Ducks of Disintegrated Lead Shot in a Simulated-Marsh Environment. J. Wildlife Diseases. 8(2):149-154.

Jarvie, A. W. P., R. N. Markall, and H.R. Potter. 1975. Chemical Alkylation of Lead. Nature. 255(5505):217-218.

Jefferies, D. J. and M. C. French. 1972. Lead Concentration in Small Mammals Trapped on Roadside Verges and Field Sites. Environ. Poll. 3(2) 147-156.

John, M. K. and C. Van Laerhoven. 1972. Lead Uptake by Lettuce and Oats as Affected by Lime, Nitrogen, and Sources of Lead. J. of Environ. Qual. 1(2):169-171.

Jones, L. H. P., C. R. Clement, and M. J. Hopper. 1973a. Lead Uptake from Solution by Perennial Ryegrass and Its Transport from Roots to Shoots. Plant and Soil. 38(3):605-619.

Jones, L. H. P., S. C. Jarvis, and D. W. Cowling. 1973b. Lead Uptake from Soils by Perennial Ryegrass and Its Relation to the Supply of an Essential Element (Sulphur). Plant and Soil. 38(3):605-619.

Karbe, L. 1972. Marine Hydroiden als Testorganismen zur Prufung der Toxizitat von Abwasserstoffen, Die Wirkung von Schwermetallen auf Kolonien von *Eirene viridula*. Marine Biol. 12(4):316-328.

Karstad, L. 1971. Angiopathy and Cardiopathy in Wild Waterfowl from Ingestion of Lead Shot. Connecticut Medicine. 35(6):355-360.

Knight, H. D. and R. G. Burau. 1973. Chronic Lead Poisoning in Horses. J. Am. Vet. Med. Assoc. 162(9):781-786.

Koeppe, D. E. And R. J. Miller. 1970. Lead Effects on Corn Mitochondrial Respiration. Science, 167(3923):1376-1377.

Lagerwerff, J. V. and A. W. Specht. 1970. Contamination of Roadside Soil and Vegetation with Cadmium, Nickel, Lead, and Zinc. Environ. Sci. Technol. 4(7):583-586.

Locke, L. N., G. E. Bagley, D. N. Frickie, and L. T. Young. 1969. Lead Poisoning and Aspergillosis in an Andean Condor. J. Am. Vet. Med. Assoc. 155(7):1052-1056.

Longcore, J. R., R. Andrews, L. N. Locke, G. E. Bagley, and L. T. Young. 1974. Toxicity of Lead and proposed Substitute Shot to Mallards. Special Scientific Report -- Wildlife No. 183, U. S. Department of the Interior, Fish and Wildlife Service, Washington, D. C., 83 pp.

Lower, W. R. 1975. Gene Frequency Differences in *D. melanogaster* Associated with Lead Smelting Operations. Sixth Annual Meeting, Environmental Mutagens Society, Miami Beach, Florida, May 9-12, 1975, pp 33-34 (abstract only).

Lu, P. Y., R. L. Metcalf, R. Furnan, R. Vogel, and J. Hassett. 1975. Model Ecosystem Studies of Lead and Cadmium and of Urban Sewage Sludge Containing these Elements. J. Environ. Qual. 4(4):505-509.

Malanchuk, J. L. and G. K. Gruendling. 1973. Toxicity of Lead Nitrate to Algae. Water, Air, Soil Poll.2(2):181-190.

Malone, C., D. E. Koeppe, and R. J. Miller. 1974. Localization of Lead Accumulation by Corn Plants. Plant Physol. 53(3):388-394.

Martin, W. E. 1972. Mercury and Lead Residues in Starlings - 1970. Pesticides Monitoring J. 6(1):27-32.

Martinez, J. D., M. Nathany, and V. Dharmarajan. 1971. Spanish Moss, a Sensor for Lead. Nature. 233(5321):564-565.

Mathis, B. J. and T. F. Cummings. 1971. Distribution of Selected Metals in Bottom Sediments, Water, Clams, Tubificid Annelids, and Fishes of the Middle Illinois River. Research Report No. 41, University of Illinois at Urbana - Champaign, Water Resources Center, Urbana Illinois,75 pp.

McIntyre, J. D. 1973. Effects of Metals on Fertilization and Development in Fish. In: Heavy Metals in the Environment, A Seminar Conducted at the Oregon State University, Water Resources Research Institute, Corvallis, Oregon, Fall Quarter, 1972, pp 163-168.

McKee, J. E. and H. W. Wolf. 1963. Water Quality Criteria. Second Edition, Publication No. 3-A., California State Water Quality Control Board, Sacramento, California, 549 pp.

Mierau, G. W. and B. E. Favara. 1975. Lead Poisoning in Roadside Populations of Deer Mice. Environ. Poll. 8(1):55-63.

Miles, C. D., J. R. Brandle, D. J. Daniel, O. Chu-Der, P. D. Schnare, and D. J. Uhlik. 1972. Inhibition of Photosystem II in Insolated Chloroplasts by Lead. Plant Physiol. 49(5):820-825.

Miller, J. E., J. Hassett, D. E. Koeppe. 1971. Accumulation and Physiological Effects of Lead in Corn. In: Trace Substances in Environmental Health-IV, Proceedings of the 4th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia, Missouri, June 23-25, 1970, D. D. Hemphill (ed.) pp 186-193.

Mitchell, R. L. and J. W. S. Reith. 1966. The Lead Content of Pasture Herbage. J. of the Sci. Food Ag. 17(10):437-440.

Monahan, T. J. 1973. Lead Inhibition of Hormotila blennista (Chlorophyceae, Chlorococcales). Phycologia. 12(3/4):247.

Morgan, G. W., F. W. Edens, P. Thaxton, and C. R. Parkhurst. 1975. Toxicity of Dietary Lead in Japanese Quail. Poultry Sci. 54(5):1636-1642.

Motto, H. L., R. H. Daines, D. M. Chilko, and C. K. Motto. 1970. Lead in Soils and Plants: Its Relationship to Traffic Volume and Proximity to Highways. Environ. Sci. Technol. 4(3):231-237.

National Academy of Sciences, Committee on Biologic Effects of Atmospheric Pollutants. 1972. Biologic Effects of Atmospheric Pollutants. Lead. Airborne Lead in Perspective, Washington, D.C. 341 pp.

Nriagu, J. O. 1972. Lead Orthophosphates. I. Solubility and Hydrolysis of Secondary Lead Orthophosphate. Inorg. Chem. 11(10):2499-2503.

Nriagu, J. O. 1973a. Lead Orthophosphates - II. Stability of Chloropyromorphite at 25 C, Geochimica et Cosmochimica Acta. 37(3):367-377.

Nriagu, J. O. 1973b. Lead Orthophosphates - III. Stabilities of Fluoropyromorphite and Bromopyromorphite at 25C. Geochimica et Cosmochimica Acta. 37(7):1735-1743.

Olson, K. W. and R. K. Skogerboe. 1975. Identification of Soil Lead Compounds from Automotive Sources. Environ. Sci. Technol. 9(3):227-230.

Page, A. L., T. J. Ganje, and M. S. Joshi. 1971. Lead Quantities in Plants, Soil, and Air Near Some Major Highways in Southern California. Hilgardia, 41(1):1-31.

Peterson, S. L., L. G. Bennett, and T. G. Tornabene. 1975. Effects of Lead on the Lipid Composition of Micrococcus luteus Cells. Appl. Microbiology. 29(5):669-679.

Price, P. W., B. J. Rathcke, and D. A. Gentry. 1974. Lead in Terrestrial Arthropods: Evidence for Biological Concentration. Environ. Entomol. 3(3):370-372.

Pringle, B. H., D. E. Hissong, E. L. Kantz, and S. T. Mulawka. 1968. Trace Metal Accumulation by Estuarine Mollusks. J. Sanitary Engineering Division, Proceedings of the American Society of Civil Engineers, 94(SA3):455-475.

Quarles, H. D., III, R. B. Hanawalt, and W. E. Odum. 1974. Lead in Small Mammals, Plants and Soil at Varying Distances from a Highway. J. Appl. Ecol. 11(3):937-947.

Rabinowitz, M. 1972. Plant Uptake of Soil and Atmospheric Lead in Southern California. Chemosphere. 1(4):175-180.

Radu, D. 1971. Action of Pb^{2+} Ions on Vegetable Cells. Farmacia. 19(10): 619-625 (abstract only).

Rains, D. W. 1971. Lead Accumulation by Wild Oats (*Arena fatua*) in a Contaminated Area. Nature. 233(5316):210-211.

Ratcliffe, J. M. 1975. An Evaluation of the Use of Biological Indicators in an Atmospheric Lead Survey. Atmos. Environ. 9(6/7):623-629.

Raymond, R. B. and R. B. Forbes. 1975. Lead in Hair of Urban and Rural Small Mammals. Bull. Environ. Contam. Toxicol. 13(5):551-553.

Rice, H. W., D. A. Leighty, and E. C. McLeod. 1973. The Effects of Some Trace Metals on Marine Phytoplankton. CRC Critical Reviews in Microbiology. 3(1):27-49.

Roberts, T. M. and G. T. Goodman. 1973. The Persistence of Heavy Metals in Soils and Natural Vegetation Following Closure of a Smelter. In: Trace Substances in Environmental Health - VII, Proceedings of the 7th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia, Missouri, June 12-14, 1973, D. D. Hemphill (ed.) pp 117-125.

Ruhling, A. and G. Tyler. 1970. Sorption and Retention of Heavy Metals in the Woodland Moss *Hylocomium splendens* (Hedw.) Br. et Sch. Oikos. 21(1): 92-97.

Santillan-Medrano, J. and J. J. Jurinak. 1975. The Chemistry of Lead and Cadmium in Soil: Solid Phase Formation, Soil Sci. Soc. of Am. Proc. 39(5):851-856.

Schlesinger, W. H. and G. L. Potter. 1974. Lead, Copper, and Cadmium Concentrations in Small Mammals in the Hubbard Brook Experimental Forest. Oikos. 24(2):148-152.

Schmitt, N., G. Brown, E. L. Devlin, A. A. Larsen, E. D. McClausland, and J. M. Saville. 1971. Lead Poisoning in Horses, An Environmental Health Hazard. Arch. Environ. Health. 23(3):185-195.

Siegel, S. M., A. Eshleman, I. Umeno, N. Puerner, and C. W. Smith. 1973. The General and Comparative Biology of Toxic Metals and Their Derivatives: Mercury and Lead. In: Mercury in the Western Environment, Proceedings of a Workshop, Portland, Oregon, February 25-26, 1971, D. R. Buhler (ed.), pp. 119-134.

Silverberg, B. A., P. T. S. Wang, and Y. K. Chaio. 1976. Ultrastructure Examination of *Aeromonas* cultured in the presence of organic lead. App. Environ. Microbiol. 11:723

Skaar, H., E. Ophus, and B. M. Gullvag. 1973. Lead Accumulation Within Nuclei of Moss Leaf Cells. Nature. 241(5386):215-216.

Smith, W. H. 1971. Lead Contamination of Roadside White Pine. Forest Sci. 17(2):195-198..98.

Smith, W. H. 1973. Metal Contamination of Urban Woody Plants. Environ. Sci. Technol. 7(7):631-636.

Smith, W. H. 1976. Lead Contamination of the Roadside Ecosystem. J. Air Poll. Control Assoc. 26(8):753-766.

Stickel, W. H. 1969. Lead Shot Poisoning of American Birds. In: Metals and Ecology Symposium, Stockholm, Sweden, March 24, 1969. Ecological Research Committee Bulletin No. 5, pp. 24-30.

Swaine, D. J. and R. L. Mitchell. 1960. Trace Element Distribution in Soil Profiles. J. of Soil Sciences. 11:347-368.

Tansy, M. F. and R. P. Roth. 1970. Pigeons: A New Role in Air Pollution. J. Air Poll. Control Assoc., 20(5):307-309.

Ter Haar, G. 1970. Air as a Source of Lead in Edible Crops. Environ. Sci. Technol., 4(3):226-229.

Ter Haar, G. 1975. Lead in The Environment--Origins, Pathways and Sinks. In: Lead, T. B. Griffin and J. H. Knelson (eds.) Environmental Quality and Safety, supplement Vol. II. Georg Thiem Publishers, Stuttgart. pp 76-94.

Ter Haar, G. L., and M. A. Bayard. 1971. Composition of Airborne Lead Particles. Nature. 232(5312):553-554. August 20.

Tidball, R. R. 1976. Lead in Soils. In: Lead in the Environment. Geological Survey Professional Paper 957, T. G. Lovering (ed.), U. S. Government Printing Office, Washington, D. C., pp 43-52.

Tornabene, T. G. and H. W. Edwards. 1972. Microbial Uptake of Lead. Science. 176(4041):1334-1335.

Tornabene, T. G. and H. W. Edwards. 1973. Effects of Lead on Bacterial Membranes. In: Trace Substances in Environmental Health - VII, Proceedings of the 7th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia, Missouri, June 12-14, 1973, D. D. Hemphill (ed.), pp. 263-266.

Tyler, G., B. Mornsjo, and B. Nilsson. 1974. Effects of Cadmium, Lead, and Sodium Salts on Nitrification in a Mull Soil. Plant and Soil. 40(1):237-242.

Ulmer, D. D. and B. L. Vallee. 1969. Effects of Lead on Biochemical Systems. In: Trace Substances in Environmental Health - II, Proceedings of the 2nd Annual Conference in Trace Substances in Environmental Health, University of Missouri, Columbia, Missouri, July 16-18, 1968, D. D. Hemphill (ed.), pp 7-27.

U. S. Department of the Interior. 1976. Use of Steel Shot for Hunting Waterfowl in the United States. U. S. Government Printing Office, Washington, D. C., 276 pp.

Watson, A. P. 1975. Trace Element Impact on Forest Floor Litter in the New Lead Belt Region of Southeastern Missouri. In: Trace Substances in Environmental Health - IX, Proceedings of the 9th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia, Missouri, June 9-12, 1975, D. D. Hemphill (ed.), pp 227-236.

Watson, A .P., R. I. Van Hook, D. R. Jackson, and D. E. Reichle. 1976. Impact of a Lead Mining-Smelting Complex on the Forest-Floor Litter Arthropod Fauna in the New Lead Belt Region of Southeast Missouri. ORNL/NSF/EATC-30, Environmental Sciences Division Publication No. 881, Oak Ridge National Laboratory, Oak Ridge Tennessee, 180 pp.

Wedding, J. B., R. W. Carlson, J. J. Stukel, and F. A. Buzzaz. 1975. erosol Deposition on Plant Leaves. Environ. Sci. Technol. 9(2):151-153.

Welch, W. R. and D. L. Dick. 1975. Lead Concentrations in Tissues of Roadside Mice. Environ. Pollution, 8(1):15-21.

Wershaw, R. L. 1976. Organic Chemistry of Lead in Natural Water Systems. In: Lead in the Environment, Geological Survey Professional Paper 957, T. G. Lovering (ed.), U. S. Government Printing Office, Washington, D. C., pp 13-16.

Wilder, D. G. 1952. The Relative Toxicity of Certain Metals to Lobsters. J. Fish. Res. Board Can. 8(7):486-487.

Wilkins, D. A. 1957. A Technique for the Measurement of Lead Tolerance in Plants. Nature. 180(4575):37-38.

Wong, P. T. S., Y. K. Chau, and P. L. Luxon. 1975. Methylation of Lead in the Environment. Nature, 253(5489):263-264.

Zimdahl, R. L. 1976. Entry and Movement in Vegetation of Lead from Air and Soil Sources. J. Air Poll. Control Assoc., 26(7): 655-660.

Zimdahl, R. L. and J. H. Arvik. 1973. Lead in Soils and Plants: A Literature Review. CRC Crit. Rev. Environ. Control. 3(2): 213-224.

Zimdahl, R. L., and J. J. Hassett. 1977. Lead in Soil. In: Lead in The Environment. W. R. Boggess, (ed.). National Science Foundation. U. S. Government Printing Office, Washington, D. C. 272 pp.

Zlochevskaya, I. V. and E. G. Rukhadze. 1968. A Study on Toxic Action of Some Lead Compounds. Microbiology. 37(6):951-955.

Zook, B. C., J. L. Carpenter, and E. B. Leeds. 1969. Lead Poisoning in Dogs. J. Amer. Vet. Med. Assoc. 155(8):1329-1342.

Zook, B. C., R. M. Sauer, and F. M. Garner. 1972. Lead Poisoning in Captive Wild Animals. J. Wildlife Diseases. 8(3): 264-272.

7.0 EFFECTS ON HUMANS

7.1 SUMMARY

The metabolism of lead is concerned with uptake and absorption, transport and distribution, and elimination.

Lead may be absorbed into the body by inhalation, ingestion, skin absorption, or placental transfer. Various studies indicate that approximately 37 - 50 percent of inhaled lead is deposited in the lungs. This amount is a function of the size and solubility of lead particles as well as rate and depth of breathing. Approximately 10 percent of ingested lead is absorbed by the gastrointestinal tract in adults. This rate is thought to be much higher in young children. Studies have also indicated that a reduction in dietary calcium increases gastrointestinal lead absorption. It appears that inorganic lead salts are not absorbed through the skin to any significant extent; however, this has not been studied extensively. Organic lead compounds, in contrast, are absorbed significantly via the skin. Placental transfer of inorganic lead has been widely reported. Exposure of the embryo or fetus to lead via placental transfer has greater potential for injury than does exposure during adulthood.

Following absorption, essentially all the lead in the body is associated with the erythrocytes; it is then distributed to various tissues throughout the body. More than 90 percent of the body burden of lead is a relatively nondiffusible fraction in the skeleton. The concentration of lead is high in hair as well as in bone, intermediate in liver, kidney and aorta, and lowest in the heart and brain. The absorption, distribution and elimination of lead results in an equilibrium state when exposure from all sources is relatively constant. In this equilibrium state, blood lead and urine lead levels fairly accurately reflect total body burden of lead. Under stable environmental conditions blood lead levels of general populations appear to be fairly stable. They tend to be higher in urban than in rural populations and in males than in females. One study has suggested that concentrations of lead in bone and in most soft tissues, such as the kidney, pancreas, and liver, rise until around the fourth or fifth decade of life and then reaches a plateau or begins to decrease. Other studies, however, have reported no such decline in the elderly.

The principal routes of elimination of lead from the body are through the feces and urine. Secondary routes are in sweat, milk, hair and desquamated skin. Fecal lead excretion is greater than excretion of lead in the urine.

The toxicological effects of lead can be viewed at three levels. First, there is the problem of sporadic episodes of acute lead intoxication; these are usually accidental or the result of ignorance of the danger of lead.

Second is the problem of clinical and subclinical intoxication of large numbers of children in urban ghetto areas who are living in pre-World War II housing. The third problem of concern in the toxicology of lead is the question of the possible harmful effects of undesirably high body stores of lead in general populations.

It has been shown that many body organs and systems are adversely affected by lead. However, much of what is known concerning these effects has been indicated only by isolated cellular or subcellular systems. The implications for the health of man are uncertain in such cases.

The critical effect of inorganic lead compounds is adverse interference with heme synthesis and, according to current knowledge, the critical organ of inorganic lead effect is hematopoietic bone marrow. Short-term inhalation of organic lead compounds results in central nervous system manifestations, suggesting that this is the critical organ of organic lead.

Lead appears to have an adverse effect on reproductive ability. Various animal studies show that parents exposed to lead had increased numbers of breeding failures, reduced litter size and weights of offspring, and reduced survival rates of offspring. Teratogenic effects of inorganic lead in animals are manifested by congenital skeletal malformations. Evidence concerning the relationship between lead and chromosomal abnormalities in humans is not definitive. Studies on chromosomal aberrations in workers professionally exposed to lead seem to favor a positive relationship.

Renal, adrenal, thyroid, prostate and pulmonary adenomas, renal adenocarcinomas and testicular carcinomas have been found in rats and mice given lead phosphate and lead acetate via dietary and parenteral routes. In contrast, epidemiological evidence to date does not indicate that lead has a similar carcinogenic effect on humans.

Lead poisoning in children is a serious but preventable problem that can be controlled in any of the three following ways: eliminating the source of lead; controlling the means by which lead enters the body; or finding and treating cases early. The sequelae of symptomatic lead poisoning include seizure disorders as well as various behavioral and functional disorders such as hyperactivity, impulsive behavior, prolonged reaction time and slowed learning ability.

The extent of the effects of lead poisoning in children is dependent on several factors. Children tend to exhibit more severe effects from lead poisoning when exposure is during a period of rapid growth. It appears that the brain is especially vulnerable during growth spurts. Age also seems to influence rate of gastrointestinal absorption of lead. In addition, both dietary components and dietary deficiencies alter gastrointestinal lead absorption rates.

Lead exerts its effects in the hematopoietic, neurologic and renal systems. Among children, renal damage associated with severe lead intoxication is frequently reversible. Permanent renal damage does occur among adults having chronic (occupational) overexposure to lead. No published data are available to suggest that renal damage occurs in asymptomatic cases.

Considerable controversy exists concerning conflicting reports of neurological damage occuring in asymptomatic or mildly symptomatic cases. Therefore, the hematopoietic system is considered the critical site for the effects of lead.

In addition to laboratory studies, and clinical studies of small groups of lead intoxicated children, a wide variety of epidemiologic studies have sought to describe and quantify the health effects of lead in various general populations. Although both gastrointestinal and respiratory absorption of lead have long been recognized as primary routes of exposure, epidemiologic studies have tended to focus upon the latter, presumable due to the relative ease with which the respiratory exposure level of populations can be characterized (based on suitable environmental monitoring data). Deriving reasonably accurate estimates of dietary lead intake has proven more difficult due to a wider range of variability among individuals with respect to daily caloric intake and composition of the diet. Thus, studies of the effects of dietary lead exposures tend to be of the laboratory type and involve intensive observation of very small groups of individuals. Moreover, past studies of metabolism of dietary lead in normal subjects have been confined almost exclusively to adult males.

Studies of the effects of respiratory lead exposure have been conducted in three general types of settings: (1) general populations, (2) groups subjected to some unusual circumstance or mode of exposure (i.e., "intermediate level" studies) and (3) studies of occupational groups. These three types of populations represent a gradient of exposure from fairly low to moderate to perhaps dangerously high.

Detailed examination of several studies of general populations is provided in the following sections. Consensus from studies of diverse general populations suggests that the contribution of air lead to blood lead is approximately 0.6 - 2.0 μg Pb/dl per 1 μg/m^3 in air. This relationship appears to hold true in a variety of communities having ambient air lead in the range from a few hundredth of 1 ppm to approximately 8-10 ppm. (See Three Cities Study, Seven Cities Study and Azar, et al., 1975, reported below.) Many interurban comparisons have failed to find significant differences in mean blood lead levels between cities or a significant correlation between air and blood lead levels within cities. Failure to establish such a relationship is possibly due to the narrow range of ambient conditions studied and/or to the relatively small contribution of air lead to total absorbed dose under the ambient conditions prevailing at the sites investigated (generally well below 5 μg/m^3). The fact that an exceptionally well done multiple regression analysis (Azar, et al., 1975) found only 43 percent of the variation in blood lead is explained by air lead supports the contention that other factors, including lead ingested from food and beverages are more important determinants of blood lead.

Studies of persons regularly exposed to unusually high ambient lead concentrations (over 10 μg/m^3 for all or part of the day) either from mobile or stationary sources have been much more successful in demonstrating relationships between levels of lead in air and those in blood. It is only under these higher dose conditions that repiratory intake accounts for an appreciable percentage of total lead absorption. Chronic exposure to intermediate levels of lead from mobile sources occurs among persons living in close proximity to dense vehicular traffic (e.g., freeways, heavily travelled urban thorofares) or from occupations which involve prolonged exposure to automotive exhaust (e.g., policemen, taxi drivers, parking garage attendants, mechanics). Populations residing near stationary sources of lead emissions such as primary and secondary smelters have also been examined. Studies conducted in each of these types of settings are reported in the text.

In general, comparisons of groups exposed to above average amounts of auto exhaust reveal mean blood lead levels somewhat higher than control groups not similarly exposed. Oftentimes, however, blood lead concentrations of the heavily exposed persons lie within, or just slightly above normal limits (with normal limits being defined as ± 2 standard deviations of the mean of some general population). Such findings are especially true of studies examining groups residing near freeways or working in traffic. Groups exposed to auto exhaust indoors, where ambient lead levels are presumably higher, consistently reveal mean blood leads 20-40 percent higher than control goups from the general population as well as a higher percentage of individuals exhibiting evidence of undue lead absorption (blood lead greater than 40 μg/dl).

Groups exposed to stationary emissions sources generally show moderate-to-marked elevations of blood lead. Moreover, in many instances, populations exhibit a gradient in blood lead levels which is inversely related to distance from the source of emissions. This type of relationship has been shown for both primary and secondary smelter areas. Several studies focus specifically on the possible effects on children living near smelting complexes. Results of investigations in El Paso, Texas; East Helena, Montana; Kellogg, Idaho; Omaha, Nebraska; Toronto (Canada) and several European countries are discussed in the following sections. Elevated blood lead (> 40 ug Pb/dl), hair lead (> 100 ppm Pb in hair), metabolic changes (increased urinary excretion of ALA or coproporphyrins), neurologic abnormalities (reduction in peripheral nerve conduction velocity) or some combination of the above were considered evidence of undue lead absorption. Children exhibited abnormalities with respect to these parameters at all of the study sites, albeit to varying degrees. Dust and dirt (in addition to airborne particulate) appear to be important sources of lead intake for children living in smelter communities.

Occupational lead exposures represent the high end of the dose-response spectrum. It is among workers who smelt, refine and manufacture lead-containing or lead-painted products that the highest and most prolonged lead exposures are found. The clinical pattern of occupational lead intoxication has changed in recent decades. Nowadays acute or fatal lead poisoning is rare; today's cases are frequently diagnosed at an earlier, asymptomatic stage based on abnormalities of biochemical, neurological, or renal function. Despite these apparent advances, however, excessive lead absorption and its attendant health effects remain a serious and all too prevalent occupational hazard in the lead industries today. Epidemiologic studies conducted between 1975 and 1976 in five major lead facilities throughout the United States showed unacceptably high blood lead levels and symtoms of lead poisoning in every plant studied (four secondary smelters and one lead chemicals plant). Hematologic, neurologic, and renal damage were consistently reported. Beyond that, this series of investigations also provided evidence of medical mismanagement of overexposed workers and poisoning of workers' children from exposure to heavily contaminated work clothing which was taken home for laundering. These and other findings from other epidemiologic studies of lead workers clearly indicate that present occupational standards and work practices do not adequately protect workers from adverse health effects.

The relationship between air lead and blood lead among industrial workers has been the subject of several investigations. These studies are reviewed in this section in terms of the policy implications their findings may have for setting more appropriate occupational standards.

The alkyl derivatives of lead are highly toxic compounds, which, like inorganic forms, may also be absorbed by inhalation or ingestion. They are readily absorbed through the skin. High solubility of alkyl lead compounds in body fluids is the basis for percutaneous toxicity whereas the volatility of these compounds underlies the hazard potential among workers engaged in the production of these compounds. Persons chronically exposed to evaporating gasoline may be at risk as well, although the requisite epidemiologic studies to quantify this potential risk have not been conducted. Because alkyl lead compounds are light sensitive and undergo rapid decomposition upon reaching the atmosphere, their presence is transient and thought to amount to less than 10 percent of inorganic lead levels. To date, no information is available on the effects of chronic, low level (non-occupational) exposure to airborne alkyl lead.

From a toxicological viewpoint, the most significant organic lead compounds are the tetraethyl and tetramethyl compounds. These compounds are used in gasoline antiknock additives; consequently potentially toxic exposures are thought to be restricted almost exclusively to the industrial sector. Because of the known occupational hazard, extensive studies have been conducted on the uptake and absorption, elimination, tissue distribution, and toxic effects of tetraethyl- and tetramethyl lead. The results of these studies are briefly summarized at the end of this chapter.

In addition to tetraethyl (TEL) and tetramethyl lead (TML), a number of miscellaneous organic lead compounds find industrial uses. These include lead soaps of organic acids such as lead stearates, tallates, naphthenates and octoates (chiefly di-2-ethylhexanoate) produced and marketed in the United States as paint driers. Any hazard encountered in the manufacture, handling and use of these products is a function of the concentration of lead actually present, the solvents contained and their activity as oxidation catalysts. Although little data are available regarding the specific toxicities of these "miscellaneous" compounds, in general, the LD_{50} values appear to be several orders of magnitude higher (less toxic) than those of TEL and TML. The behavior of these lead soaps in biological systems also appears more similar to inorganic lead than to that of TEL and TML.

7.2 BIOLOGICAL PATHWAYS

7.2.1 Uptake and Absorption

Lead may be taken into the body by inhalation, ingestion, skin absorption, and placental transfer.

7.2.1.1 Inhalation--

The uptake of any inhaled particulate material is a function of its size and solubility. When inhaled, the small lead particles (less than 1 to 2 micrometers in diameter) tend to be retained by the respiratory tract to be absorbed or coughed up and swallowed later. Particles of hygroscopic material are deposited in the lungs in a higher percentage than non-hygroscopic ones because as they take up water from the moist air in the respiratory tract their size increases. Deposition of inhaled particles will also vary with rate and depth of breathing. Minimum deposition occurs during rapid, shallow conditions of respiration (Nozaki, 1966). Kehoe (1961 a, b; 1964) found that the deposition of lead sesquioxide particles in the lung ranged from 35 percent of the amount inhaled when the mean particle diameter was 0.05 micrometer, up to 54 percent for a diameter of 0.075 micrometer. When the mean diameter was approximately 1 micrometer, retention was 43 to 53 percent. Kehoe (1961 a-d) estimated that 40 percent of the lead (as lead sesquioxide) deposited in the airways were transferred to the gastrointestinal tract. This transfer probably involved particles approximately 2.9 μm mass median equivalent diameter (MMED) and larger (National Academy of Science, 1972). In a study of lead retention in the lungs of lead workers and ship breakers, Mehani (1966) found that 37 to 47 percent by weight of the inspired lead was retained in the lungs of the lead exposed workers. These concentrations varied with severity of effort at work, (that is, with respiratory rate), size of particles including those of 3 micrometers, and the number of particles being retained in the mouth and throat and either expectorated or swallowed and excreted in the feces. In this case, 50 percent of the lead particles were greater than 2.9 micrometers (MMED). It is believed that the particles in this range of diameter were transferred to the gastrointestinal tract.

Rabinowitz et al., (1974) calculated that the quantity of lead absorbed from a typical urban atmosphere (lead concentration equals 1 to 2 $\mu g/m^3$) is 15 ± 3 μg per day.

7.2.1.2 Ingestion--

The absorption of lead in the intestine depends upon age, chemical form, dietary calcium level, Vitamin D intake, diet composition, quantity consumed, and gastric acidity. Other studies have shown that dietary iron, copper, selenium, and ascorbic acid levels also influence lead absorption. Vitamin D in the diet increases the concentration of lead in the blood and in the femur (Sobel, et al., 1938). Reduction of dietary calcium increases the absorption of lead. Kostial (1971a) found that lead retention was 1.4 times lower in 5 to 7 day old rats fed cow's milk with calcium and phosphate additives than in those fed pure cow's milk. Six and Goyer (1972) showed that lowering dietary lead also increases lead absorption.

Net absorption of lead from the gastrointestinal tract has been found to be quite low in those species which have been studied, and the percentage absorbed does not appear to be proportional to the dose. In laboratory animals, the amount of lead absorbed from the gastrointestinal tract appears to be much less than that in man. Net absorption of lead in sheep is approximately 1.3 percent over a range of 2 to 108 milligrams Pb/day and is quite similar for rabbits (Blaxter, 1950). The form in which lead is administered to animals does not appear to have a major effect on its absorption. Calves, poisoned with a series of lead salts, showed little difference in uptake when the lead was given as the phosphate, oxide or carbonate, or even as dried flakes. Only galena and metallic lead seemed to be absorbed poorly (Allcroft, 1950).

Generally, the uptake of lead from the gastrointestinal tract is less complete than it is from the lungs, assuming that the amount of lead absorbed from the lungs is equivalent to the amount deposited (that is, 37 - 40 percent of the amount inhaled). Experimental studies have revealed that gastrointestinal absorption of lead varies with age. Kehoe (1961a) found that, in adults, absorption of lead from the gastrointestinal tract is approximately 10 percent of the amount ingested. (See Table 7.1). Alexander et al., (1973) reported that in children, aged 3 months to 8 1/2 years, 53 percent of ingested lead is absorbed by the gastrointestinal tract with 18 percent retention. More recently, Ziegler, et al., (1978) found that absorption averaged 42 percent in a group of infants and toddlers.

Rabinowitz, et al., (1975) used stable isotope and balance studies to investigate the human uptake of dietary and atmospheric lead. Five healthy volunteers were maintained in a hospital metabolic unit for up to 6 months in order to assess absorption, excretion, pool sizes and fluxes of lead under typical urban conditions. Subjects were fed constant low-lead diets, supplemented with non-radioactive isotope tracers. The concentration of tracer lead and total lead in diet, feces, urine, blood, hair, nails, sweat, bile, gastric and pancreatic secretions and bone were measured by mass spectrometry.

Alimentary absorption of both the tracer lead nitrate and food lead varied from 6-14 percent in these subjects, while in rats as high as 70 per cent absorption was observed when tracer was administered during fasting. The data on the kinetics of tracer lead within the body could be adequately described by a 3-compartment model. By transferrring 3 subjects to a room with filtered air and observing the rate at which their blood lead fell, it was determined that about 15 µg/day of lead is inspired, about one-half as much as originates in diet. This was confirmed by the lead balance and the incomplete labelling to blood by a dietary tracer.

TABLE 7.1 LEAD INGESTION AND EXCRETION OF A NORMAL HUMAN SUBJECT[a]

8-Week Periods	Lead Ingested, mg	Lead Excreted, mg Total	In Feces	In Urine	Net Change in Body Lead, mg
1st	13.59	11.95	10.45	1.50	+1.64
2nd	13.31	15.13	13.63	1.50	-1.82
3rd	13.16	13.82	12.45	1.37	-0.66
4th	11.51	11.59	10.41	1.18	-0.08
5th	9.30	8.93	7.88	1.05	+0.37
6th	9.24	9.05	8.16	0.89	+0.19
7th	12.75	13.74	12.86	0.88	-0.99
Subtotal	82.86	84.21	75.84	8.37	-1.35
8th	22.21	17.61	15.85	1.76	+4.60
9th	18.17	15.05	13.76	1.29	+3.12
Subtotal	40.38	32.66	29.61	3.05	+7.72
Total	123.24	116.87	105.45	11.42	+6.37

[a] Source: Kehoe (1961a-d). Reprinted from Journal Royal Institute Public Health Hygiene.

7.2.1.3 Skin Absorption--

Penetration of inorganic lead through the intact skin has not been studied extensively. Limited data indicate that inorganic salts of lead are not absorbed to any significant extent. (Laug and Kunze, 1948).

Hamilton and Hardy (1974) reported a case of skin absorption of inorganic lead from the application to the skin of a theatrical grease paint containing 40 percent lead oxide. Conceivably, the lead oxide might have been converted to a liquid-soluble organic salt. Lead soaps, for example, naphthenate, used in industrial lubricants, however, are absorbed significantly via skin (Hine et al., 1969).

Rastogi and Clausen (1976) studied the absorption of lead through the skin by comparing the effect produced by lead naphthenate or lead acetate solution, when coated on the skin of rats, with data obtained from subcutaneous injections of these solutions. Body weight and liver size and weight decreased in rats receiving the subcutaneous dose. δ-Aminolevulinic acid dehydrase (ALA-D) in liver was decreased in all rats that were treated with lead compounds. The distribution of absorbed lead was evaluated by assay of the lead content in brain, liver, kidney, spleen and muscle in the rats. (Table 7.2). The results of this study seem to verify that absorption of lead through the skin does occur. Also, the decline in ALA-D activity was either equal to or more pronounced when lead naphthenate was administered than when lead acetate was administered; however, the lead naphthenate produced lower lead concentrations in the liver than did the acetate compound. These findings seem to indicate that lead naphthenate is comparatively more toxic than lead acetate.

In a study of the toxicity of lead naphthenate, Van Peteghem and DeVos (1974) observed that 8 percent of the 102 employees having occupational contact with lead-containing oils showed a blood lead content of over 40 μg/dl and also had an increase in δ-amino levulinic acid excretion in the urine. However, the observed increases were all below the maximum acceptable concentration for adults.

7.2.1.4 Placental Transfer--

The inorganic forms of lead have a greater potential for injury to the embryo, fetus, neonate and child than to the mature adult. The fetus seems most susceptible to the effects of lead during the phase of rapid growth and it has been postulated that fetal tissues may be able to concentrate lead at least during the first 16 weeks of gestation (Karlag and Moeller, 1958). Cord blood was found to have lead concentrations approximating those found in maternal blood (Scanlon, 1971, Harris and Holley, 1972, and Rajegowda et al., 1972). Scanlon has found lead concentrations as high as 39 μg/dl in the umbilical cord. The blood of newborns contains lead in concentrations similar to that in cord blood (Robinson, et al., 1958).

In a study on normal humans, Barltrop (1969) observed that placental transfer of lead began as early as the twelfth week of gestation and that the

TABLE 7.2 ALA-D ACTIVITY IN LIVER AND DISTRIBUTION OF LEAD IN VARIOUS ORGANS IN RATS[a]

Experimental Group	Mean ALA-D Activity[b] Parts catalyzed/g liver	Lead Content in μg/g Wet Tissue[c]				
		Brain	Kidney	Liver	Spleen	Muscle
Control	619 ± 24	11.2 ± 1.7	7.9 ± 2.3	5.0 ± 0.8	8.8 ± 1.4	2.9 ± 0.5
Cutaneous lead acetate	544 ± 35[d]	13.0 ± 1.8	55.4 ± 5.2[d]	18.8 ± 2.5[d]	8.9 ± 1.2	7.7 ± 2.0[d]
Cutaneous lead naphthenate	524 ± 18[d]	12.8 ± 1.6	34.8 ± 3.2[d]	9.6 ± 0.8[d]	8.8 ± 1.1	11.4 ± 2.4[d]
Subcutaneous lead acetate	473 ± 19[d]	15.8 ± 1.8[d]	321.0 ± 18.0[d]	108.0 ± 10.0[d]	81.9 ± 6.8[d]	17.8 ± 2.2[d]
Subcutaneous lead naphthenate	473 ± 24[d]	21.3 ± 2.6[d]	184.0 ± 7.7[d]	56.3 ± 6.6[d]	50.6 ± 2.2[d]	25.2 ± 2.1[d]

[a] Source: Adapted from Rastogi and Clausen (1976). Reprinted, with permission from Toxicology.(c) Elsevier - North Holland Publishers, Inc., Amsterdam (1976).

[b] Assayed in triplicate.

[c] Assayed in duplicate.

[d] P <0.001.

lead content in fetal tissues increased throughout pregnancy. Fetal bone was found to have the highest tissue-lead level (80 μg/g). Lower lead levels were found in the placenta, heart, kidney, liver, and blood. The total amount of lead transferred per day during pregnancy appeared to be less than 303 micrograms.

Finklea et al., (1972) found that lead is bound to and crosses the placenta, with fetal blood levels being lower than maternal levels, and with erythrocyte levels showing a greater difference than whole blood. According to Baumslag (1975), transplacental transfer might not occur in mothers with sufficiently high copper levels (quantity not given) as copper may be protective.

Kostial and Momcilovic (1974), investigated the transfer of lead during various stages of gestation and lactation to determine the period of maximal transfer from mother to offspring. Tracer doses of lead-203 were given intravenously to rats on the 18th day of pregnancy and on the 4th and 15th day of lactation. Calcium-47 was given at the same time to relate the results of lead transfer to the transfer of calcium to offspring. The transplacental transport of lead was eight times lower than that of calcium while transmammary transport of lead was four times lower.

Chaube, et al., (1972) in a study of human embryonic and fetal lead concentrations, detected lead in two-thirds of the first trimester specimens without relation to age. Fifty embryos and fetuses ranging from 31 to 261 gestational age were used. In fetal tissues, lead was found in 77, 15, and 30 percent, respectively, of the liver, brain, and kidney samples analyzed. In the embryos the concentration of lead ranged from 0.38 to 2.0 micrograms per gram of wet tissue weight. Only two fetuses had detectable lead in the brain. In the liver, the concentration ranged from 0.84 to 4.04 micrograms per gram of wet tissue weight, and in the kidney from 0.9 to 2.3 micrograms per gram of wet tissue weight.

Haas, et al., (1973), reporting on 294 sets of maternal and fetal blood samples, found lead levels in maternal samples were 16.9 ± 8.6 μg/dl; fetal lead levels were 15.0 ± 7.9 μg/dl. The coefficient of correlation of blood levels in the newborn with levels in their mothers equaled 0.57.

Schroeder and Tipton (1968) found lead in significant concentrations in young children of several nationalities, even in stillbirths, indicating considerable placental transfer (see Table 7.3).

7.2.2 Transport and Distribution

Following absorption, essentially all the lead in the body is associated with the erythrocytes. It is then distributed to various tissues with concentrations being highest in bone and hair, intermediate in liver, kidney, and aorta, and lowest in heart and brain. Schroeder and Tipton (1968) have observed that under conditions approximating steady state, more than 90 percent of the lead resides as a relatively nondiffusible fraction in the skeleton. The body tends to reject lead at a rate commensurate with the rate of ingestion.

TABLE 7.3 **TISSUE LEAD CONCENTRATION IN CHILDREN AND ADOLESCENTS**[a]

Place of Origin	N	Aorta	Liver	Kidney	Pancreas	Lung	Testis	Heart	Brain	Spleen	Bone
		Mean Lead Concentration, ppm ash									
America											
Stillborn	22	65	53	38	-	18	-	25	10	37	-
0-19	23	24	74	62	31	18	9	5	0	10	2
Africa	5	-	100	39	18	23	-	0	13	-	-
India	9	82	100	90	44	68	76	33	10	50	10
Japan	3	27	64	98	26	34	72	15	13	23	27

[a] Source: Schroeder and Tipton. Reprinted with permission from Archives Environmental Health. (c) American Medical Association, 1968.

N = Number of samples.

Therefore, the biological fluid levels (blood and urine) fluctuate with the amount of ingested lead. Since only about 10 percent of the ingested lead is absorbed into the body, lead concentrations in the feces reflect short-term fluctuations in the lead ingested while the urine and blood levels are more indicative of lead concentrations over long periods of time.

Lead is associated with the erythrocytes or the plasma proteins, and a small quantity is in the free, ionized state. Approximately 95 percent of the lead in the circulating blood is attached to the red cell. The rate of lead uptake from the plasma by the erythrocytes is temperature dependent. Clarkson and Kench (1958), on reexamination of Mortensen and Kellog's data (1944), showed that the ratio of the velocities of uptake at 10 and 20 C was given by the equation:

$$k\ 20/k = 5.61 \times 10^4/2.16 \times 10^4 \text{ minutes},$$

thus $Q_{10} = 2.8$, and

$$\text{energy of activation} = 15{,}800 \text{ cal/mole}.$$

Since lipid-free stroma does not bind lead effectively, the lipids and lipid proteins in the cell membrane seem to act as a stong binding site for lead (Jung, 1947 and Vincent, 1958, cited in Waldron and Stöffen, 1974; Teisinger, et al., 1958). The fact that lead is bound to plasma proteins is not surprising since other heavy metals like mercury and cadmium also show a strong affinity for ligands such as phosphates, cysteinyl and histidyl side chains of proteins, purines, pteridines, and porphyrins (Vallee and Ulmer, 1972). Possibilities suggested for the manner in which lead is bound to the erythrocyte include a peptized lead phosphate sol (Clarkson and Kench, 1958), a colloidal lead phosphate (Aub, et al., 1925), a diglyceryl phosphate (Maxwell and Bischoff, 1929a,b) mixed salts with calcium and chloride (Jowett, 1932), and a protein-phenolic complex (Reddi, 1953). The affinity of lead toward sulfhydryl (SH) groups suggests a series of reactions on the membrane which lead to a S-Pb-S complex (Passow and Tillman, 1955, cited in Waldron and Stöffen, 1974).

Barltrop and Smith (1971) have reexamined the lead-binding properties of human erythrocytes and investigated the cell fraction in which binding occurs. Their results suggest that lead is bound to the cell contents rather than to stromal material. This was confirmed by the absence of significant binding of lead when washed stroma were tested in place of hemolyzed erythrocytes. The observed interaction with the prehemoglobin band was attributed to residual hemoglobin bound to that fraction. Comparison of the elution characteristics of this band with a calibration graph indicated a mean molecular weight of 240,000. Stromal proteins extracted from red cell ghosts have been shown to have molecular weights ranging from 10,000 to 170,000; however, these values are unlikely to apply to material obtained by simple hemolysis. The lack of correspondence between hemoglobin concentration and lead binding in fractions obtained from the Sephadex column at elution volumes of 350 milliliters and above suggests that there by be some low-molecular-weight material in addition to hemoglobin that interacted with lead.

Lead disappears from the blood at a rate which indicates first-order reaction kinetics and may be the sum of three exponential functions (Castellino and Aloj, 1964). Stover (1959) concluded that lead rapidly transferred to the extravasular spaces from the plasma as the latter level declined. To maintain dynamic equilibrium between red-cell and plasma lead, on the one hand, and extracellular and intracellular lead on the other, it is suggested that the ionic fraction of the plasma lead is transferred slowly to other body compartments. A similar rationale applies to the lead concentration in urine, with blood showing a somewhat lesser fluctuation than urine (Kehoe, 1961a-d). The concentration of lead in the blood of the general population is fairly stable. The lead content in the blood does not change significantly with age, neither among North Americans (U.S. Department of Health Education and Welfare, 1965) nor among some other nationalities (Horiuchi, 1970, cited in TGOMA, 1973; Horiuchi and Takada, 1954, and Horiuchi, et al., 1959, cited in National Academy of Sciences (1972). There was no increase in the concentration of lead in serum with age in U.S. residents (Butt, et al., 1964). Coulston (1973) and Knelson, et al., (1973) described a study in which 20 human volunteers were exposed to 10.9 μg/m^3 of lead sesquioxide 23 hours per day for 4 months (see Figure 7.1).

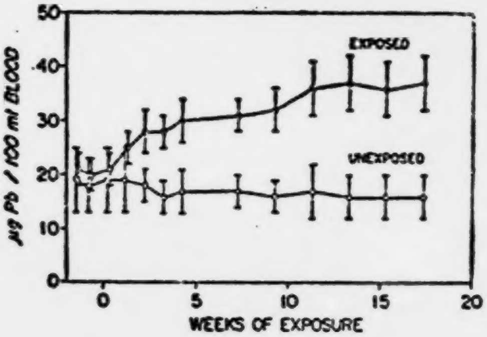


Figure 7.1 Levels of blood lead in volunteers exposed to 10.9 micrograms per cublic meter lead sesquioxide.
Source: Coulston. Reprinted with permission from Environmental Quality and Safety, Vol. 2., F. Coulston and F. Korte (eds.), (c) Academic Press, Inc., 1973.

In the exposed group the blood lead concentration increased to nearly twice their preexposure levels while the level remained relatively unchanged in the controls. During the final 6 weeks of the experiment, there was no apparent increase in the blood lead of the exposed men, the level remaining at about 35 μg/dl. After about 6 weeks of exposure the ALAD activity had decreased to about one-half the preexposure value in the men.

Goldwater and Hoover (1967) determined blood-lead levels of persons in 16 different countries. A total of 801 blood specimens were analyzed for lead content using the dithizone method. In general, urban means were higher than rural means. There appeared to be no statistically significant differences between countries with respect to geographical location or degree of industrialization. The blood lead level of their study was 17 μg/dl of blood with a standard deviation of 11 μg/dl.

Kubota, et al., (1968) who made a detailed investigation of blood-lead levels of male residents of 19 cities in the continental United States, found that the means from one city to another varied widely as did individual values within each city. Earlier, Hofreuter, et al., (1961) observed variations among residents in Los Angeles with respect to both sex and urbanization. Blood lead levels were significantly higher in males as compared to females and in rural as compared to urban inhabitants.

Extensive studies of soft-tissue lead distrubution have been reported by Schroeder and Tipton (1968), Schroeder and Balassa, (1961), Tipton and Cook (1963), Tipton and Shafer (1964), and Tipton et al., (1965). Barry and Mossman (1970) confirm these earlier data. Table 7.4 shows the variations in lead concentration with geographical orgin. The brain and the heart show consistently low values. The authors provide no explanation for the geographic variation in these values other than to point out the fact that most of the values are highest for the most urbanized/industrialized areas. In general the aorta has the highest concentration followed by the liver and kidney.

Schroeder and Balassa (1961) and Schroeder and Tipton (1968) found that concentrations of lead in the soft tissue, for example, kidney, pancreas, liver, lung, and aorta increase with age, the rise being particularly sharp in the aorta. These values continue to rise until approximately age 40-50 (age 60-70 for the aorta), at which point they begin to level off. Age-linked accumulations of lead in tissues of Americans were also found for larynx, trachea, and prostate (Schroeder and Balassa, 1961). In aortas collected at autopsy from 18 male residents of Baltimore the highest aortic levels of lead were found in those persons 38 years or older (Poklis, 1975). All but two of these subjects had dense atheromas present. The lead content of the trachea at autopsy from 17 Baltimore residents sampled in 1972 ranged from 0.6 to 8.8 micrograms per gram of fresh tissue (Poklis and Freimuth, 1975). Contrary to the observation of Schroeder and Tipton (1968), no significant correlation of tracheal lead and age was demonstrated by these data. The mean tracheal lead concentration of the samples collected in 1972 was compared with the mean lead concentration of Baltimore residents reported in 1957. From the increase in trachea lead, it may be concluded that from 1957 to 1972 there was an increase in the lead "body burden" of Baltimore residents. While the increase may not have been sufficient to affect public health of Baltimore residents, it does reflect an increase in lead exposure in the Baltimore environment.

Bone contains approximately 90 percent of the total body burden of lead, which is deposited in the form of the insoluble tertiary lead phosphate. Data by Schroeder and Balassa (1961) and Schroeder and Tipton (1968) indicate that the concentration of lead in the bones rises steadily to about the 4th decade

TABLE 7.4 VARIATION IN HUMAN TISSUE LEAD CONCENTRATION WITH GEOGRAPHICAL LOCATION[a]

Place of origin	N	Aorta	Liver	Kidney	Pancreas	Lung	Testis	Heart	Brain	Spleen	Bone
		Mean Lead Concentration, ppm ash									
Nine American cities	150	140	130	98	49	47	12	5	5	27	43
San Francisco	27	220	160	54	65	38	20	10	5	44	-
Switzerland	9	32	59	45	42	32	23	14	5	20	-
Africa	54	71	64	36	24	28	29	5	5	21	-
Middle East	37	140	70	62	34	42	32	24	14	40	26
Far East	74	91	97	63	39	43	35	19	10	33	30

[a] Source: Schroeder and Tipton. Reprinted with permission from Archives Environmental Health. (c) American Medical Association, 1968.

and remains relatively constant and then decreases in the 7th or 8th decade. However, the results reported by Schroeder and Tipton were based on only 3 individuals, and additional confirmation of this conclusion appears needed.

Barry and Mossman (1970) and Barry (1973) concluded that there is no decrease in bone lead with old age and suggested that the drop reported by some of the earlier investigators was due to the small number of samples. This confirms earlier work by Morris (1940), who in calculating regression coefficients between lead concentration in femur, ribs, and vertebrae, and age found that lead contents rose significantly and gave correlation coefficients equal to 0.48, 0.36, and 0.28, respectively. He concluded that more lead was stored in the long bones than in either ribs or the vertebrae, however, there does not seem to be general agreement as to which bone contains the greatest concentration of lead. Holtzman (1963) demonstrated that the concentration of lead-210 was higher in trabecular than in cortical bone. The lead in the skull bones seemed to be most representative of the total skeleton since their concentration deviated least from the mean. Rib concentrations deviated most from the mean and concentrations in the tibia were very low.

Gross, et al., (1975) assayed 29 tissues per individual from autopsies of 46 white males ranging in age from 20 to 84 years (see Table 7.5) and found the following: 43 had blood-lead concentrations of 40 μg/dl or less; one had a blood lead concentration of 41 μg/dl, one a concentration of 43 μg/dl and one had a concentration of 106.5 μg/dl. On a wet-weight basis (see Table 7.6), calcified tissues (bones and aorta) contained the highest concentration of lead; the glandular tissues (liver, kidney, and pancreas) were intermediate; and the remaining tissues which were mostly of a nonglandular nature had the lowest concentrations. Table 7.6 shows the distribution of lead in the bones and aorta. The low lead values for the bones of the individual in the 5th decade may reflect a chance selection of individuals with low lifetime lead exposures. A rapid increase of lead in the aorta was found after the 5th decade.

Gross and Pfitzer (1975) found that concentrations of more than 2 micrograms of lead per gram in the aorta were associated with severe atheroclerosis involving calcium deposits. As reflected in the skeletal lead, the overall body content of lead increased with age, but the concentrations in many soft tissues did not change and in several tissues lead concentrations decreased with age.

Sumino, et al., (1975) determined the amounts of 15 heavy metals in 15 female cadavers (average weight 55 kilograms, average age 39 years.) The highest level of lead was found in the adrenal glands (1.2 plus or minus 0.79 ppm). The average lead content of the female tissue samples was higher than that in corresponding male tissue samples except in the liver. Significant differences were found in small intestines ($p = 0.01$), lung, brain, adrenal glands, and spleen ($p = 0.05$).

Table 7.5 TISSUES RANKED ON THE BASIS OF OVERALL MEAN CONCENTRATIONS OF LEAD[a,b]
ppm, wet-weight basis

Tissue	N	X/SD	r	Tissue	N	X/SD	r
Tibia	45	14.09/11.22	0.53[c]	Thyroid	40	0.17/0.11	0.30
Skull	44	13.81/9.09	0.50[c]	Testes	43	0.15/0.09	-0.26
Rib	45	7.14/4.19	0.38[d]	Jejunum	45	0.12/0.05	-0.30[d]
Vertebrae	44	4.42/2.52	0.21	Brain, grey	39	0.11/0.05	-0.18
Aorta	45	1.65/1.82	0.52[c]	Brain, white	41	0.11/0.04	-0.09
Liver	45	0.98/0.53	-0.34[d]	Fascia	43	0.11/0.05	-0.05
Nodes	41	0.86/0.93	0.15	Stomach	45	0.10/0.05	-0.32
Kid. cor.	45	0.79/0.42	-0.53[c]	Bladder	43	0.09/0.04	-0.02
Kid. med.	44	0.48/0.22	-0.38[d]	Skin	43	0.08/0.03	-0.11
Pancreas	45	0.46/0.23	-0.45[c]	Heart	43	0.08/0.04	-0.12
Spleen	43	0.33/0.15	-0.02	Cecum	45	0.07/0.03	-0.07
Lung	42	0.36/0.12	0.13	Muscle	44	0.07/0.03	-0.23
Adrenal	41	0.25/0.12	-0.38[d]	Urine	8	0.06/0.11	0.24
Blood	43	0.21/0.09	-0.29	Adipose	43	0.04/0.01	0.11
Prostate	41	0.20/0.22	-0.08				

[a] Source: Gross, et al. Reprinted with permission from Toxicology and Applied Pharmacology. (c) Academic Press, Inc., 1975.

[b] Shown are number of tissues (N), their means ±SD and correlation of tissue concentrations vs age.

[c] $p < 0.01$.

[d] $p < 0.05$.

N = Number of samples.

TABLE 7.6 AGE-RELATED INCREASES OF LEAD CONCENTRATION IN BODY TISSUES[a,b] (ppm, wet-weight basis)

Tissue	Decade, years					
	20-29	30-39	40-49	50-59	60-69	70-79
Tibia	6.61±0.92 (6)	9.37±4.64 (4)	10.77±5.09 (8)	9.68±4.43 (9)	18.57±8.34 (11)	28.36±21.37 (6)
Skull	7.27±1.76 (6)	9.07±4.31 (4)	12.85±5.12 (8)	10.13±4.21 (9)	19.60±8.17 (11)	21.76±18.06 (5)
Rib	4.06±1.92 (6)	5.48±3.82 (4)	8.04±4.13 (8)	4.83±1.55 (9)	9.77±4.36 (11)	9.42±4.65 (6)
Vertebrae	3.48±1.50 (6)	2.98±1.84 (4)	4.98±1.52 (8)	2.88±1.20 (9)	6.66±3.24 (11)	4.17±2.26 (6)
Aorta	0.40±0.18 (6)	0.86±0.71 (4)	0.75±0.16 (8)	1.14±0.46 (9)	2.83±2.55 (11)	3.32±2.18 (6)

[a] Source: Gross, et al. Reprinted with permission from Toxicology and Applied Pharmacology. (c) Academic Press, Inc. 1975.

[b] Shown are the decade means ±SD. The number of tissues in each decade are shown in parentheses.

7.2.3 Elimination

The principal routes of elimination of lead from the body are through the feces and urine. Secondary routes include sweat, milk, hair, and discarded and desquamated skin. Lead concentrations of approximately 0.01 ppm have been found in the milk of normal cows. This seems toxicologically insignificant (Hammond and Aronson, 1964). In experimental animals, lead is excreted into the bile, and injected lead may be found in the feces. For example, Blaxter and Cowie (1946) after administration of lead acetate to sheep, found that 7.5 percent of the dose was excreted in 6 days of which 81 percent was in the bile.

The classical studies of Kehoe (1961a-d) indicate that under normal conditions not more than 10 percent of ingested lead is absorbed by adults. However, under abnormal conditions the excretion of inorganic lead is enhanced by procedures or conditions that favor mobilization from bone and soft-tissue burdens.

The excretion of lead into the urine is probably not as great as excretion in the bile. The excretion of lead through the kidney is hampered by the interaction of lead and heavy metals in general with ligands present in erythrocytes and/or plasma proteins. Vostal (1963 and 1966), cited in TGOMA (1973) and Vostal and Heller (1968) investigated lead excretion in man and dogs and found that the renal tubule reabsorbed a constant amount of lead, with the effect being pH-dependent, increasing at low pH and decreasing or entirely disappearing at high pH. More recently these same investigators found that in the chicken, lead passes into the urine not only by glomerular filtration, but also by direct transport across the tubular wall.

Kehoe's work (1961a-d) indicates that in conditions approximating a steady-state in which oral input roughly parallels urinary and fecal output, the urinary excretion is about 10 percent of the oral input and the fecal output is close to 90 percent. Most of the lead in the feces represents lead which has passed unabsorbed through the gut. The contribution to total excretion of inhaled lead was not determined, a factor which would have likely elevated the estimated proportion of the total lead intake excreted via the urine.

The estimates of half-time for lead in bone have varied from 64 days in the spine of rats (Torvik, et al., 1974) to 7,500 days in the skeleton of a dog (Fisher, 1969). The half-time varies with different bones, presumably due to the relative proportions of cortical and trabecular bone. The International Commission on Radiological Protection (ICRP) has used 10 years as the biological half-life for lead in bones of humans (ICRP, 1959). Following a single dose of ^{210}Pb in dogs, disappearance of the label from blood was followed for 280 days. The biological half-life of lead in the body was estimated to be 1940 days (Hursh, 1973).

7.3 TOXIC EFFECTS

The present-day environmental problem of lead may be viewed at three levels. First, there is the problem of sporadic episodes of acute lead intoxication. These are largely accidental or the result of ignorance of the danger of lead. Second is the problem of clinical and subclinical intoxication of large numbers of children in urban ghetto areas who are living in pre-World War II housing. This is largely a socioeconomic problem but corrective measures are possible; perhaps the most promising is increased monitoring and surveillance for early detection and treatment (see Section 9.3.3). The third aspect of the environmental problem concerns the question of the possible harmful effects of body stores of lead in general populations.

An answer to this latter question depends on increased knowledge of the biologic effects of lead, particularly regarding the effect of trace quantities on cellular function (Goyer, 1971).

Many organs and systems have been shown to be adversely affected by lead. However, much of what is known concerning effects has been adduced only in isolated cellular or subcellular systems. The implications for the health of man are uncertain in such cases. The subject of enzymatic and cellular responses to lead and organ system toxicities is a complex topic, which would require a much more detailed discussion than is possible in this document. Information on this topic can be found in the recent report on the environmental impacts of lead (Bell, et al., 1978) which treats this topic in detail, including discussions of renal, hematopoietic, nervous system, immune system, cardiovascular system, and endocrine system toxicities and the factors influencing or modifying toxicity.

In this report, discussion is restricted to representative acute toxicities, the occupational standards derived from them and the toxic effects on the reproductive system.

7.3.1 Toxicities of Inorganic Lead Compounds

Representative toxicities and occupational standards for industrial inorganic lead compounds are shown in Table 7.7 (as noted earlier, inorganic compounds are considered to include all those where a carbon atom is not directly connected to lead; this includes the lead salts of organic acids which are used as paint driers, etc.).

U.S. Occupational Standards include those promulgated by the Occupational Safety and Health Administration; those shown are time-weighted averages (TWA), based on continuous exposure for 8-hour day and a 40-hour work week, and are stated in terms of air lead concentrations (as lead). Also shown are the TLV's (Threshold Limit Values) established by the American Conference of Govermental Industrial Hygienists (ACGIH), which issues recommended air lead concentrations, also time-weighted averages based on a 40-hour work week.

TABLE 7.7 REPRESENTATIVE TOXICITIES AND OCCUPATIONAL STANDARDS FOR SEVEN COMMERCIALLY IMPORTANT INORGANIC LEAD COMPOUNDS[a]

Compound and Chemical Formula	Standards, $\mu g/m^3$		Toxicities, mg/kg[f]		
	U.S. Occupational[b]	TLV[c,d]			
Lead arsenate $PbHASO_4$ or $Pb_3(AsO_4)_2$	150	150	oral, rat oral, human	LD_{50} LD_{Lo}	100 1.4
Lead chromate, basic $PbO \cdot PbCrO_4$	200	150	ipr, guinea pig scu, rat	LD_{50} TD_{Lo}	400 150
Lead dioxide PbO_2	200				
Lead monoxide (litharge) PbO	200		ipr rat	LD_{Lo}	430
Lead tetraoxide (red lead) Pb_3O_4	200		ipr rat ipr rat	LD_{Lo} LD_{50}	1,500 200
Lead naphthenate $C_nH_{2n}-2O_2$ $C_nH_{2n}-4O_2$ $C_nH_{2n}-6O_2$	—		oral, rat skn rat	LD_{50} LD_{50}	5,100 520
Lead stearate (approx) $Pb(C_{18}H_{35}O_2)_2$	--		oral, guinea pig	LD_{Lo}	6000 mg/kg
Inorganic lead compounds (all)	50[e]				

TABLE 7.7 (Continued)

Notes:

[a]Source: Registry of Toxic Effects of Chemical Substances (NIOSH, 1977).
Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment (ACGIH, 1977)
U.S. Department of Labor (1978).

[b]U.S. Occupational Standard (OSHA) based on time-weighted average (TWA) for continuous exposure (8-hr day and 40-hr week).

[c]Threshold Limit Value - based on time - weighted average for continuous exposure 8-hr day and 40-hr week (American Conference of Governmental Industrial Hygienists (1977).

[d]TLV of 150 $\mu g/m^3$ applies to fumes and dusts of all inorganic lead compounds.

[e]Revised standard for all inorganic lead compounds (U.S. Department of Labor, 1978).

[f]Toxicity data descriptors:

1. routes of administration:
   ipr - intraperitoneal
   scu - subcutaneous
   skn - dermal or cutaneous (intact skin)
   ivn - intravenous
   par - parenteral, unspecified.
   inh - inhalation

2. toxic response terms:
   LD_{50} - Lethal Dose Fifty - A calculated dose of a chemical substance which is expected to cause the death of 50 percent of an entire defined experimental animal population, as determined from the exposure to the substance, by any route other than inhalation, of a significant number from that population.

   LD_{Lo} - Lethal Dose Low - the lowest dose of a substance other than LD_{50} introduced by any route other than inhalation over any given period of time and reported to have caused death in humans or animals when introduced in one or more divided portions.

   TD_{Lo} - Toxic Dose Low - the lowest dose of a substance introduced by any route other than inhalation over any given period of time and reported to produce any toxic effect in humans or to produce carcinogenic, teratogenic, mutagenic, or neoplastigenic effects in humans or animals.

   MLD - Minimum Lethal Dose.

   LD_{50} - Lethal Concentration Fifty - a calculated concentration of a substance in air, exposure to which for a specified length of time would cause the death of 50 percent of an entire defined experimental animal population as determined from the exposure to the substance of a significant number from that population.

OSHA has promulgated new regulations which establish the maximum allowable concentration for lead and its inorganic compounds in work room air at 50 $\mu g/m^3$ (U.S. Department of Labor, 1978).

For inorganic lead compounds, the critical effect of lead is adverse interference with heme synthesis and according to present knowledge, the critical organ of lead effect is the hematopoietic bone marrow. For organic lead compounds and, specifically, the alkyllead compounds, short-term inhalation first results in central nervous system manifestations, suggesting that this is the critical organ for organic lead. (See Section 7.6.4).

Lead salts of organic acids would be expected to show critical effects more characteristic of inorganic lead compounds than alkylleads, although absorption through the skin might lead to neurological manifestations. The solubility of the lead compounds in tissue fluids is of prime importance (more important than the physical nature - dust, fumes, mists, or vapors) in determining their passage to the critical organs.

Generally the toxicity of the lead salts of organic acids (lead naphthenate, lead tallate, lead stearate, and lead-2-ethylhexanoate) is associated with (1) hydrolysis or decomposition products, resulting in inorganic lead compound and (2) solvent systems employed in their commercial utilization, which influence their absorption. Thus, the migration of lead/lead compounds from polymeric films might result in increased toxicity of lead.

7.3.2 Reproductive Effects

The effects of lead on human reproductive functions per se have received little study in recent years. Consequently, much of what appears in the literature concerning human reproductive effects comes from very old studies of workers exposed to levels of lead orders of magnitude higher than today's lead workers, or alternatively from rather unscientific foreign reports of similar age. Lancranjan's (1975) study is the only recent investigation focusing exclusively on human fertility effects (specifically spermatogenic abnormalities) under reasonable well defined levels of lead exposure. Some cell culture studies of mutational activity of lead are also available.

7.3.2.1 Fertility Reduction--

When both male and female rats and mice were exposed to 25 micrograms of lead (as lead acetate) per liter of drinking water, the offspring had increased numbers of breeding failures, runts, young deaths and dead litters (Schroeder, 1973). Unspecified inorganic lead salts administered to pregnant mice and rats in low doses (25 ppm) in drinking water produced runting, reproductive failure and shortened life spans (Schroeder and Mitchener, 1971).

Hilderbrand et al., (1973) investigated the effect of lead acetate on reproduction and metabolism in rats. Eighty sexually mature males and eighty sexually mature females were maintained in a controlled environment at a constant temperature of 25.5 C. The males and females were divided into three groups with each group containing 20 rats. Group 1 served as controls; group 2 was treated orally with 5 micrograms of lead acetate for 30 days; and group 3 was treated with 100 micrograms of lead acetate for 30 days. These dosages of lead were presumably administered daily, although the authors do not specifically state. Results of this investigation revealed the following:

1) In males, when the lead concentration in the blood increased from 14 micrograms per 100 milliliters to 26 μg/dl, impotence and prostatic hyperplasia resulted.
2) When the lead level increased to 50 μg/dl, testicular damage occurred and spermatogenesis was inhibited.
3) In the female, when lead concentration in blood increased from 14 μg/dl to 30 μg/dl, irregularity of estrus occurred.
4) When lead levels reached 30 μg/dl, persistent vaginal estrus occurred after normal estrus, and the development of ovarian follicular systs with a reduction in the number of corpora lutea was noted.
5) No toxicity or mortality occurred in the treated animals.

Stowe and Goyer (1971), also have observed that the reproductive capacity of male rats is reduced as a result of lead exposures. The paternally-transmitted effect includes reduction of litter size, weights of offsprings and, reduced survival rates of the offsprings. Goyer and Rhyne (1973) reported that lead workers having blood-lead levels equal to 62 to 88 μg/dl displayed gap-break type aberrations of chromatids similar to to those reported in animal studies by Muro and Goyer, (1969). A variety of nonspecific changes in chromosome morphology (adhesions and spiralizing defects), and increases in tetraploid mitoses and the mitotic index were also noted.

Lancranjan, et al., (1975) investigated the reproductive ability of 150 men occupationally exposed to lead at a storage battery plant. One hundred of these men had a range of occupational exposure to lead of 1 to 23 years (8.5 mean), and 50 were technicians and office workers who worked in annex workrooms in a lead-polluted environment for 1 to 27 (mean 6) years. Lead-poisoned workmen (23) had mean values of lead in blood of 74.5 ± 26 μg/dl; workmen with moderately increased absorption (42), 52.8 ± 21 μg/dl; slightly increased absorption (35), 41 ± 12 μg/dl; physiologic absorption (50), 23 ± 14 μg/dl. Workmen having lead absorption ranging from slight to high exhibited a substantial increase in spermatogenic abnormalities (asthenospermia, hypospermia, or oligospermia and/or teratospermia). Because tests for hypothalamo-pituitary influence were negative, it appeared that effects on the testes were direct.

7.3.2.2 Mutagenicity--

Nuclear polyploidy and abnormalities in mitosis resulting from lead toxicity have been found in bone marrow cells by several investigators. Chromosomes from leukocyte cultures from mice fed a diet containing 1 percent lead acetate showed an increased number of gap-break-type aberrations (Muro and Goyer, 1969). The observed chromosomal abnormalities largely involved only single chromatids. Failure to have a paired defect (break or gap) in both chromatids of a chromosome suggested that damage occurred after the deoxyribonucleic acid (DNA) synthetic phase of the cell cycle. There is a possible relationship between increased DNA'ase activity and chromosome damage as a marked increase in deoxyribonuclease activity has been demonstrated in urine from lead-poisoned rats (Muro and Goyer, 1969).

Studies on chromosomal aberrations in workers professionally exposed to lead, lead and some of its compounds, and mixtures of lead, cadmium, zinc, and tin, still do not answer the question whether lead can cause chromosomal abnormalities. At best, evidence seems to be in favor of there being a causal relationship. Whereas Bauchinger et al., (1972), O'Riordan and Evans (1974), Schmid et al., (1972), and Speding et al., (1972) could not detect any severe chromosomal aberrations in workers from different lead industries, Bauchinger et al., (1976), De Knudt et al., (1973), Forni and Secchi (1972), Forni et al., (1976), and Schwanitz et al., (1970, 1975) observed a significant increase in such aberrations. De Knudt (1977) concludes that lead is not the only factor responsible for the severe aberrations found in some workers in lead industries and that it can even be surmised that other factors must be considered, for example diet, photochemical effects, and intoxication with other heavy metals (cadmium, zinc).

7.3.2.3 Teratogenicity--

Although effects of lead on reproductive fitness have been surmised to occur in individuals with increased body burden of lead, this effect has not been clarified. Hamilton and Hardy (1974) reported that lead poisoning in women industrial workers in the early 1900's resulted in decreased fertility and an increased abortion rate. Exposure of women to concentrations of lead prevalent in industry at that time no longer happens. Consequently, the effect of lead on the human endocrine and reproductive systems received relatively little attention in comparison with studies on the hematopoietic, renal, and nervous systems. Observations concerning the adverse effects of lead on animals and man are herein discussed.

Stowe and Goyer (1971) found that lead is toxic to gametes of both male and female rats, resulting in decreased numbers and size of offspring from poisoned rats. Vermande-Van Eck and Meigs (1960) observed histopathological changes in the ovaries of lead-poisoned rhesus monkeys. Such changes have not been observed in humans. Ferm and Carpenter (1967) were able to induce specific congenital-skeletal malformations in golden hamster embryos. On either day 7, 8, or 9 of pregnancy various salts of lead (acetate, chloride, nitrate) were injected into the mother through the lingual vein. The dosage was 50 mg/kg made up in distilled water and the volume of the injection was 1 ml/100 g of body weight. The embryos were recovered between day 12 and day 15 of the normal 16-day gestation period. The use of lead nitrate on 3 different days of gestation shows that the 8-day old embryo seems most sensitive to the effect of this compound. The malformations caused by each of the 3 lead compounds were primarily localized within the developing sacral and tail vartebrae and were characterized by varying degree of tail malformations.

Ferm (1969) examined the teratogenic effects of both cadmium and lead when injected separately into pregnant hamsters. On the eighth day of gestation the hamsters were anesthetized with pentobarbital and injected with distilled water (controls), cadmium sulfate (2 mg Cd/kg), lead acetate (25 or 50 mg Pb/kg) or combinations of cadmium sulfate and lead acetate. The frequency and severity of clefts in the lip and palate usually caused by cadmium were reduced in the presence of lead, while the posterior tail malformations caused by lead appeared to be potentiated in the presence of cadmium (Ferm, 1969). The possibility is suggested that in the case of tail bud malformation, cadmium and lead interact additively on certain enzymes, but that lead blocks the effect of cadmium on the differentiating visceral arch system, thus preventing the facial abnormalities.

Lead acetate injected intravenously into pregnant prairie moles, Microtus ochrogaster, during organogenic stages was teratogenic at 32 mg/kg, but not at 8 or 16 mg/kg (Kruckenberg, et al., 1976). Total litter resorption occurred only at doses of 64 mg/kg. No histopathologic changes, such as eosinophilic intranuclear inclusion bodies in the proximal convoluted tubules of the kidney, were observed in treated pregnant females.

Palmisano, et al., (1969) attributed a case of neuromuscular abnormalities and growth impairment of a child to lead poisoning as a result of the consumption by the pregnant mother of illicit whiskey.

Summarizing, the teratogenic effect of lead needs additional clarification and research.

7.3.3 Carcinogenicity

In contrast to man, rodents are susceptible to lead-induced cancers. Renal, adrenal, thyroid, prostate, and pulmonary adenomas, renal adenocarcinomas, and testicular carcinomas have been observed in rats and mice which had received lead phosphate, lead acetate, and basic lead acetate via dietary and/or parenteral routes (Sunderman, 1971). Sunderman also reported lymphomas resulting from the subcutaneous injection of lead.

Oyasu, et al., (1970) found that cerebral gliomas (usually poorly differentiated, malignant tumors) were induced in rats with dietary lead subacetate and/or 2-acetylaminofluorene (AAF). The highest incidence (8.6 percent) of gliomas was in rats ingesting dietary lead subacetate. With AAF the glioma incidence was 2.5 percent while in the controls it was 0.3 percent. An increased incidence of lung cancer has been reported in lead, copper and zinc smelter workers, and in the residents of communities surrounding smelters (Anonymous, 1975a). The report adds that arsenic emitted from these plants may be the actual cause of cancer.

A followup study of 425 workers exposed to lead in a storage battery factory was conducted by Dingwall-Fordyce and Lane (1963). They reported "no evidence to suggest that malignant disease was associated with lead absorption". Similarly, a recent epidemological study of causes of death among U. S. lead workers indicated that although the deaths from malignant neoplasms were slightly higher than expected, the incidence was not elevated to the point of statistical significance (Cooper and Gaffey, 1975).

Martell (1975) postulates that small, insoluble particles of radioactive lead (lead-210) are absorbed by tobacco plants as they grow. The radioactive lead is inhaled in the lungs with tobacco smoke, where it gives off alpha rays that destroy the lung cells, or the radioactive lead particles spread throughout the body, and may cause some bone, liver and stomach cancers.

Summarizing, the research to date indicates that cancer cannot be singled out as a particular risk for individuals with chronic lead poisoning.

7.4 LEAD POISONING IN CHILDREN

Lead poisoning is a preventable disease that generally can be controlled in three ways: by eliminating the sources of lead; by controlling the means by which lead enters the body; or by finding and treating cases early. Unless the source of lead poisoning in a child's environment is eliminated or contained following diagnosis and treatment, recurrence of lead poisoning is probable. Thus, early diagnosis must be coupled with efforts to identify and remove the sources of poisoning. The issue of leaded paint (specifically, the issue of what constitutes a safe level of lead in paint) has been addressed in a major report by the National Academy of Sciences (1976). In this report it is noted that answer to the lead in paint question presupposes answers to three preliminary questions, namely:

1) What are the adverse effects of lead?
2) What dose of lead is required to produce adverse effects? and
3) What is the estimated daily intake of lead in a child with pica for paint.

Although it is recognized that the total amount of lead assimilated may be derived from a variety of environmental sources, the NAS report is concerned mainly with the absorption of lead from the ingestion of lead-containing paints by young children.

7.4.1 Adverse Effects of Lead in Children

According to National Academy of Sciences (1976) there are basically three stages in childhood lead poisoning:

1) Asymptomatic lead poisoning in which no clinical symptoms are apparent, but in which measurable metabolic changes occur,
2) Symptomatic lead poisoning in which clinical symptoms such as anorexia, vomiting, apathy, atoxia, drowsiness or irritability occur, and
3) Lead encephalopathy with cerebral edema, in which coma or convulsions occur.

The sequelae of lead encephalopathy, noted by the NAS panel, include seizure disorders, severe mental retardation and death. Likewise, the sequalae of symptomatic but less severe lead poisoning include seizure disorders as well as various behavioral and functional disorders, usually captioned under minimal brain dysfunction. Clinical studies indicate that the latter syndrome may include hyperactivity, impulsive behavior, prolonged reaction time, perceptual disorders, and slowed learning ability. Minimal brain dysfunction might also result from asymptomatic lead poisoning. Significantly, the sequelae in each diagnostic category of lead poisoning do not necessarily occur in every child with a diagnosis; each individual is unique in his response.

The NAS panel concluded that the question of deciding on which organ system is the most sensitive, i.e., defining the "critical effect" is very important when setting "safe levels" of lead. Studies of both hematologic effects and neurologic effects show varying degrees of response with equivalent exposure to lead. For example, hematologic effects appear in some 1 - 5 year old children when blood lead levels reach 30 - 40 μg/dl, while an increased frequency of neurologic effects have been shown only in those children in the range of 50 - 60 μg/dl or above.

Since the effects of lead in the hematopoietic system are reversible, they do not constitute sequelae. Lead interferes with the formation of hemoglobin at several stages, it reduces the life span of the red blood cells which results in lead induced anemia. In encephalopathy, acute renal injury (Fanconi syndrome) may also occur. In children this is usually reversibile, assuming that the child is removed from the source of lead which produced the toxicity in the first place

7.4.2 Lead Dose Necessary to Produce Adverse Effects

In the context of this discussion "dose" may mean (1) the quantity of lead administered, (2) the quantity absorbed, or (3) the quantity present in the affected organs, tissues or biological fluids. External dose refers to the amount of lead entering the body. The internal dose represents the amount of lead present in the organs, tissues, or biological fluids. Herein, blood lead concentrations are used as indicators of internal dose.

Because individual variability influences the estimate of a dose necessary to produce an adverse effect, any estimate of a safe dose level should allow a margin of safety for highly susceptible individuals who are affected by relatively low doses (National Academy of Sciences,1976). This estimate can be obtained from results of population surveys which measure both dose and effect in each individual and may be expressed as dose-response curves. This relationship is expressed in Figure 7.2, where blood lead is used as an indicator of internal dose and erythrocyte protoporphyrin as an indicator of effect. Positive reactors are those individuals who show an effect greater than the anticipated mean plus two standard deviations. On plotting the percentage of positive reactors for each blood lead group, it can be seen that highly susceptible individual groups show a positive response at relatively low blood lead levels, while highly resistant individuals show a normal response at relatively high blood lead levels.

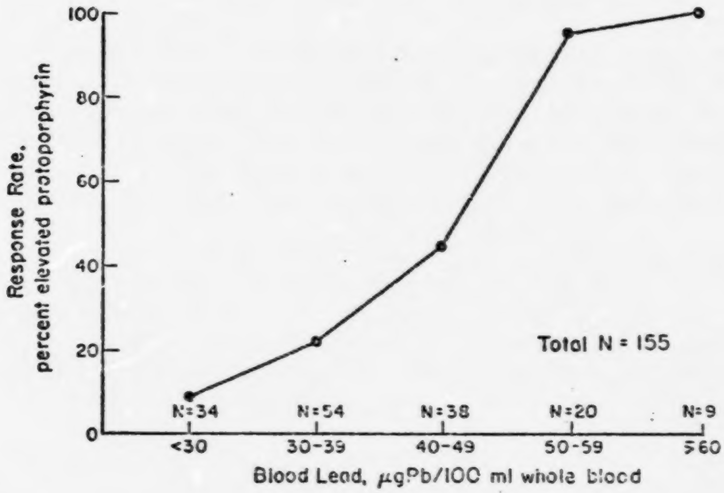


Figure 7.2 Dose-response relationship for the effects of internal doses of lead as Pb-B on erythrocyte protoporphyrin.

Source: Chisolm (1977).
Reprinted from Clinical Chemistry (1977).

Zielhuis (1975a and 1975b) also plotted dose-response curves for each measure of lead's effect to obtain a series of dose-response relationships. As the NAS panel stated, while dose-response information is helpful in defining safe levels of toxic substances, other parameters must be considered. These are individual variability, age, and diet.

Individual variability influences the estimate of a dose required to produce an adverse effect. In a heterogeneous population, some members seem to be affected by comparatively low doses of lead, while other individuals will show little or no effect at high doses. Generally, however, the percentage of individuals in a population who show a specific effect will be dose-sensitive. This condition must be recognized in identifying safe dose levels for the entire population under investigation.

Evidence in both animals and humans indicates that both age and diet are primary factors influencing the absorption and effects of lead (National Academy of Sciences, 1976). Dobbing and Sands (1973) report that the growth spurt of the human brain begins during midpregnancy. They conclude that (1) glial replication and differentiation extends to at least the end of the first postnatal year and quite likely beyond 18 months, (2) myelination continues into the third and fourth years and (3) cerebellar growth is most rapid during the first 18 months of postnatal life. About 83 percent of the human brain growth spurt is postnatal (Dobbing, 1974). Studies of malnutrition in both experimental animals and human infants have demonstrated the vulnerability of the developing brain. Hyponutrition occurring during the growth spurt of the brain of the rat produced a permanent reduction in both body and brain weight as well as behavioral changes (Dobbing and Sands, 1973). Hyponutrition before this period had less severe effect on the central nervous system (CNS) development.

The growth spurt in rats occurs during the first 25 days after birth along with glial cell multiplication during the first half of this period. Rapid myelination, dendritic authorization, synaptic connections, dramatic metabolic and neurochemical development and rapid cerebral growth occur during this period. Permanent clumsiness associated with cerebral deficits caused by hyponutrition occur during the growth spurt (Dobbing, 1974).

Studies of malnutrition in human infants also have shown that the brain is especially vulnerable during the growth spurt. Pyloritis occurs between birth and 3 months of age, is surgically correctable and is not associated with any particular socioeconomic or cultural group (Klein, et al., 1975). Reduced I.Q. levels were found in school-age boys suffering from malnutrition during the first two years of life (Hertzig, et al., 1972).

Age also seems to alter the intestinal absorption rate for lead. It has been hypothesized that gastrointestinal absorption of lead is more efficient in children than in adults. Various animal studies support this position as does a study by Alexander et al. (1973) involving children aged 3 months to 8 $^{1}/2$ years.

A more recent study leaves little doubt that a higher percentage of gastrointestinal lead intake is absorbed in children than in adults (Ziegler, et al., 1978). Twelve normal infants aged 14 to 746 days served as subjects for this study. Eighty-nine metabolic balance studies were performed to determine lead intake and fecal and urinary excretion. Net absorption and retention were calculated from the results of these studies. Urinary excretion increased with increasing intake; however, it amounted to only a small portion of total excretion. Lead intake and fecal excretion were significantly correlated. Absorption, calculated as lead intake minus fecal excretion and expressed as a percentage of intake, averaged 26.2 percent. Average lead retention was 11.3 percent, expressed as a percentage of intake. Both absorption and retention of lead were significantly correlated with intake.

Ziegler, et al, analyzed the results of balance studies with intakes greater than 5 μg/kg/day separately, since it is likely that most children have intakes higher than 5 μg/kg/day. Average net absorption was 42 percent of intake, and net retention averaged 32 percent of intake when intakes were greater than 5 μg/kg/day. Fecal excretion of lead exceeded intake in 7 of 28 studies with intake less than 5 μg/kg/day. This was true in only 3 of 61 studies with intakes higher than this value. Studies have shown that gastrointestinal absorption of lead in adults is approximately 10 percent of intake (Kehoe, 1961a; Rabinowitz et al., 1975). Therefore, Ziegler's work lends substantial weight to the hypothesis that gastrointestinal absorption of lead is more efficient in children than in adults.

Both dietary components and dietary deficiencies alter the intestinal absorption of lead (National Academy of Sciences, 1976). In experimental animals, the intestinal absorption of lead is significantly increased if lead is administered in oils, fats, or milk rather than in a dry diet. Similar studies are not available, nor possible, in young children. Dietary deficiencies such as calcium, copper, and iron increase the absorption of lead in rats. Dietary deficiencies of calcium and, especially, iron have been reported to be prevalent among preschool age children, especially those in the lower socioeconomic groups. Because of the rapid growth rate during early childhood, iron reserves may be marginal even in apparently healthy children. An additional risk factor, pica, the repetitive ingestion of non-food substances, occurs in at least 50 percent of preschool children between 12 and 36 months of age (National Academy of Sciences, 1976).

Summarily, several factors make the young child less resistant to lower levels of lead than the adult. Pica may lead to ingestion of lead-containing paint chips; the young age makes the child susceptible to lead-induced neurologic damage; and both age and diet may elevate the intestinal absorption rate for lead (National Academy of Sciences, 1976).

Lead exerts its effects in the hematopoietic, neurologic, and renal systems. Renal damage is a reversible effect observed in severe cases and no published data are available to suggest that damage occurs in asymptomatic cases. Considerable controversy exists concerning conflicting reports of neurologic damage occurring in asymptomatic or mildly symptomatic cases. At least until this argument is resolved, the hematopoietic system is considered the critical site for lead's effects. Thus, using blood lead as a measure of the internal dose of lead, different effects can be observed as blood lead levels increase. Lead-induced anemia has been found in both adults and children. Lead causes multiple interferences in the formation of hemoglobin (National Academy of Sciences, 1972), including inhibition of the enzymes, δ-aminolevulinic acid dehyratase (ALA-D), and ferrochelatase (Pentschew and Garro, 1966). The inhibition of these enzymes results in an accumulation of δ-aminolevulinic acid in urine (ALA-U) and free erythrocyte protoporphyrin (FEP) in blood. It is now known that zinc protoporphyrin and not the free protoporphyrin IX is present in excess in the circulating erythrocytes in lead poisoning and iron deficiency (Lamola and Yamane, 1974). Population studies of children in the United States have rarely included enough children with <20 μg Pb/dl to determine this lower threshold level.

A second threshold is observed in children when blood lead levels reach 35 - 40 μg/dl (Chisolm, et al., 1975; Kammholz, et al., 1972, Piomelli, et al., 1973, and Sassa, et al., 1973). When blood lead levels reach 40 - 50 μg/dl, the excretion of ALA-U begins to rise in both adults and children (Selander and Cramer, 1970, and Tola, et al., 1973). Pueschel, et al., (1972) found a significant negative relationship between hemoglobin and blood lead levels in children. Blood lead levels > 60 μg/dl were almost always associated with hemoglobin levels <10 g/dl.Betts, et al., (1973) found hemoglobin levels < 11 g/dl in 36 percent of children with 37-56μg Pb/dl, 71 percent with 60 - 100 μg Pb /dl, and 89 percent with ≥ 100 μg Pb/dl. Rosen and Trinidad (1974) found a negative relationship between hematocrit and blood lead concentrations in children at levels exceeding 40 μg/dl.

Subtle neurological effects are difficult to measure in both adults and children and even more difficult to attribute to a single cause such as lead poisoning. There is currently no set of neurochemical tests for measuring changes that is comparable to tests available for measuring changes in the hematopoietic system. Present measurements of neurologic changes accomplished through the use of functional tests such as I.Q. tests. Confounding factors such as parental I.Q., parental education level, socioeconomic status, and birth trauma also affect the results of these tests. Studies aiming to show a relationship between I.Q. and exposure to lead should include control/study matches for age, birth rank, parental I.Q., socioeconomic status, nutrition, and pica. In addtion, there should be studies in which the presence or absence of exposure to lead in the early years is well documented. Many reported studies lack a correct experimental design, a proper control group, and firm documentation concerning lead exposure.

The studies of de la Burde and Choate (1972 and 1975) meet most of the criteria of a prospective study. Both study and control children came from an on-going Child Development Study at the Medical College of Virginia in Richmond. Mothers were followed during pregnancy and delivery and children followed for eight postnatal years. The study group consisted of 67 asymptomatic children who had a positive history of pica for paint or plaster, lived in deteriorated old housing, had positive urinary coproporphyrin tests and either a blood lead level >40 μg Pb/dl or blood lead >30 μg Pb/dl and positive radiographic findings for lead lines in the long bones. We feel that this combination of criteria for selecting the study group was more reliable than a selection based on blood lead levels alone. Even so, the absence of serial blood levels, which were not feasible at the time, is the major weakness of this study. This weakness is largely overcome by dependence on x-rays (Betts et al., 1973) and positive urine coproporphyrin tests (Benson and Chisolm, 1960) are generally associated with blood lead concentrations equal to or greater than 60 μg Pb/dl. Lead levels in shed deciduous teeth were determined several years later on teeth from 29 of the lead-exposed children and 32 of the control children. The mean tooth lead level for the study group was significantly higher than the mean tooth lead level of the control group. The control group consisted of 70 children who had a negative history of pica for paint or plaster, lived in modern housing, did not visit older housing for day care and had negative tests for coproporphyrin in urine. In addition, all children were excluded from both groups who showed neurologic abnormalities or developmental lag either during the newborn period or at four months, if abnormalities were noted on the Bayley scale at eight months, or if confirmed or suspected disease of the central nervous system was noted anytime before seven years of age. In addition, the groups were comparable in age, sex, race, mother's non-verbal I.Q., socio-economic status, family composition and possible sources of family upheaval such as death in the family, foster home placement or working mother.

Neurological and psychological tests were administered to both groups at four years of age and again at seven years of age. Fifty-eight children from each group also had tests repeated at eight years of age. At four years of age, the most significant differences between the groups were in the areas of fine motor coordination and behavior. Failure on fine motor tests occurred almost twice as frequently in the lead-exposed group as in the control group. Deviation in overall behavior ratings occurred almost three times as frequently in the lead-exposed group. Mean I.Q. scores, as measured by the Stanford-Binet test, were 89 ± 13.1 for the lead-exposed group and 94 ± 10.5 for the control group. At seven years of age, neurologic examination revealed deficits in more than twice as many children from the study group as from the control group. Full-scale I.Q., as measured on the Wechsler Intelligence Scale for Children revealed that the majority of children from both groups had average intelligence, although the mean I.Q.'s were statistically significantly ($p < 0.01$) lower in the lead-exposed group. The frequency of results in the borderline or mentally defective range was higher in the lead-exposed group. Short attention span and minimal goal orientation occurred in 32 percent of lead-exposed children and 14 percent of control children. Poor academic progress was noted in 27.8 percent of lead-exposed children and 4.1 percent of control children. The number of children repeating at least one grade was higher in the lead-exposed group (25.9 percent) than in the control group (6.1 percent). Eleven lead-exposed children and four control children were receiving speech therapy for speech impediments.

De la Burde and Choate (1972, 1975) felt that the most significant difference between the groups was in the area of behavior and that this was the primary cause for poor school performance. Among the lead-exposed group, five had been seen by psychiatrists, one had been institutionalized and three were subject to seizures. None of these findings occurred in any of the control children. A review of school records revealed that hyperactivity, explosive behavior and frequent temper tantrums occurred in 19 lead-exposed children and 5 control children. The behavior problems which had been apparent at four years of age, but which were adequately handled in the home environment, persisted at seven years and prevented appropriate functioning in the school environment.

The results of several additional studies indicate a relationship between increased lead absorption and neurologic deficits in young children. These include the studies by Albert, et al., (1974), Perino and Ernhart (1974), Beattie, et al., (1975), and David et al., (1972).

Although no single study cited above represents a perfect model to detect subtle neurologic differences between groups, an increased frequency of irreversible neurobehavioral deficits seems to exist in those children with blood lead levels in the range of 50 - 60 µg Pb/dl or above. Reversible neurobehavioral effects appear at somewhat lower blood levels. However, it is not clear as to whether the exposure levels of 50 - 60 µg/dl or above represent the levels which were responsible for the behavioral deficits observed.

7.4.3 Estimated Lead Intake in a Child with Pica for Paint

The National Bureau of Standards has published the results of mathematical models together with the assumptions and data used to formulate the models (Gilsinn, 1972). From this model it was estimated that approximately 600,000 children in 241 standard metropolitan statistical areas (SMSA'S) had elevated blood lead levels (>40 µg/dl). However, as Goyer and Mehlman (1977) point out, at the present time the models and assumptions are only partially validated. The study has also been criticized for using only data from the east and midwest. (See also Section 8.6.3)

The incidence of lead poisoning is highest among one-to-five year old children who live in run-down urban neighborhoods where peeling paint and broken plaster often prevail (Pueschel, et al., 1972). A relatively small number of cases result from exposure to improperly glazed pottery or exposure to industrial sources, (e.g., smelters and battery factories). The mechanism by which lead industries contribute to childhood lead poisoning can be direct, as in the case of lead emissions to ambient air, and fallout of particulate lead causing elevation of lead levels in dust and dirt, and indirect, such as household contamination due to lead carried into the house on leadworker's clothing.

Even in the absence of significant industrial sources, the preschool-age child is exposed to multiple sources of lead, including dust, canned foods and liquids, and paint. It is the cumulative intake and absorption from these various sources that is important. Inhalation of average urban air (1 - 3 µg Pb/m^3 is considered insignificant in comparison with the above sources.

Paint provides the most concentrated source of lead potentially available to a young child. Dried house paint films containing 0.5 percent lead would provide 5,000 µg Pb/g paint. Comparison of model x-ray films of known quantities of paint with abdominal x-ray films taken of children known to have pica for paint indicated 7 of 10 randomly selected films showed radiopacities equivalent to an estimated one gram of paint consumed by each child (King and Schaplowsky, 1974).

Since house dust lead levels in newer inner city houses were significantly lower than in the older inner city houses (2 - 24 µg Pb/square foot compared with 33 - 456 µg Pb/square foot floor surface, (Sayre, et al., 1974) concluded that the source of lead probably originated from the powdering of old lead paints. From a study of dirt samples around painted frame farm houses remote from traffic Ter Haar and Aronow (1974) concluded that lead paint was the principle cause of elevated soil lead levels and that lead from fallout dust was an insignificant source of lead intake in the children studied.

Although wide variations in lead intake could occur as a result of the parents' choice of food for their child, an estimate of 93 ± 36 μg Pb/day was obtained from the analysis of the dietary intake of 333 infants aged 1 to 12 months. (National Canners Association study, cited by Kolbye, et al., 1974). This estimate corresponds to about half the total adult dietary intake. Studies by Alexander, et al., (1973) in 11 healthy children receiving a normal diet showed a mean lead intake of 10.6 μg Pb/kg body weight per day and a mean fecal lead output of 5.1 μg/kg/day. In Alexander's studies average daily intake and outputs are calculated on a per kilogram basis for an average two-year old child weighing 12.5 kilograms. Obviously in Kolbye's studies the mean lead intake per kilogram of body weight was somewhat higher. Since Kolbye did not determine the fecal lead output the percent of lead absorbed cannot be calculated.

However, from Alexander's studies it is estimated that 50 per cent of lead from foods is absorbed by a young child. This estimate will be used in this discussion on the hazard of 0.5 percent lead paint. Studies in animals indicate that lead compounds, incorporated into a paint matrix, are absorbed only one-fourth to one-half as well as the free lead salts. (Gage and Litchfield, 1969). Using an average of one-third for estimating the child's absorption of lead from paint, experimental data indicate that children will absorb one-third of 50 percent, or an average of 17 percent of the lead from paint.

Table 7.8 (National Academy of Sciences, 1976) gives the estimated amounts of lead absorbed and excreted, based on an absorption factor and estimates of weekly intakes of 1, 2, or 3 grams of 0.5 percent paint. Average daily intakes are also calculated on a per kilogram basis for an average two-year old child weighing 12.5 kilograms.

Table 7.8 CALCULATED LEAD INTAKE AND ABSORBED DOSE FROM PAINT PICA[a]

Paint Ingested Per Week, g	Calculated Lead Intake[b] μg/day	μg/kg/day	Lead Absorbed[c] μg/day	μg/kg/day	Lead Excreted[d], μg/day
1	714	56.1	121	9.7	593
2	1,429	114.3	243	19.4	1,186
3	2,143	171.4	354	29.1	1,779

[a] Source: National Academy of Sciences, (1976)
[b] Assumes paint contains 0.5 pecent lead
[c] Absorption estimated at 17 percent
[d] Excretion in feces estimated at 83 percent

Thus, from Table 7.8, a 12.5 kg child consuming 1 gram of 0.5 percent lead paint per week would have a daily intake of 57.1 µg Pb/kg body weight, a fivefold increase above that found in a normal diet. Two grams of paint would produce an eleven-fold increase, and three grams, a sixteen-fold increase.

Clinical evidence indicates that children with pica are apt to ingest one to three grams of paint per week (National Academy of Sciences, 1976). Since the ingestion of between one and two grams of 0.5 percent lead paint per week would be enough to produce daily fecal outputs equivalent to those found in children with >60 µg Pb/dl of blood, the NAS panel concluded a level of 0.5 percent lead in paint cannot be considered a "safe level".* The above method for determining the hazard of lead paint is premised on relating lead intake to the appearance of early clinical illness and significant risk of central nervous system (CNS) effects.

A second method relates lead intake to blood lead levels associated with the appearance of metabolic effects in children. The average blood lead level in normal children is approximately 20 µg Pb/dl (Lepow, et al., 1974). Early metabolic changes in the hematologic system start when blood lead levels reach the range of 30 to 40 µg/dl. Thus, it would be desirable to insure that mean blood lead levels for groups do not exceed 20 µg/dl. Table 7.9 (National Academy of Sciences, 1976) shows the effect of the ingestion of 0.5 per lead paint on blood lead levels.

TABLE 7.9 CALCULATED DAILY EXTERNAL DOSE AND ASSOCIATED INTERNAL DOSE[a]

Increase in Blood Lead, µg/dl	Lead Absorbed Each Day, µg/kg/day	Corresponding Lead Intake, µg/kg/day[b]	Total Lead Intake Necessary For 10 kg Child, µg/day	12.5 kg Child, µg/day
17	1.43	8.41	84.1	105.1
34	2.86	16.82	168.2	210.3

[a] Source: National Academy of Sciences, (1976)
[b] Assumes 17 percent absorption.

---

*It may be pointed out that lead paints up until very recently contained much more than 0.5 percent lead; the paints originally applied to now-substandard housing may have contained as much as 60 - 65 percent lead.

The ingestion of 16.8 mg paint/day or 33.6 mg paint per day containing 0.5 percent lead would raise blood levels by 17 µg/dl or 34 µg/dl in a 10 kg child. Similarly, a 12.5 kg child would need to ingest either 21 mg paint/day or 42.6 mg paint/day. It is not unusual for a child with pica for paint to consume more than 1,000 mg paint per week or 143 mg paint per day (King and Schaplowsky, 1974). This second method also shows that a level of 0.5 per cent lead in paint clearly represents a hazard to a child with pica for paint. However, because many factors modify lead intake, absorption rates, and individual susceptibility, the foregoing two methods used for determining the hazard of 0.5 percent lead paint, cannot be considered suitable for application to every child. Factors (discussed earlier) such as age, diet, and nutrition affect the increase or decrease of lead absorption by one individual. Only after these parameters are resolved can we hope to get additional clarification of the hematopoietic, neurological, and renal effects of lead and thereby define a safer daily permissible intake for all groups.

However, with respect to the dose calculated by the NAS panel for a child with pica (Table 7.8), at the new upper limit of 0.06 percent lead in paints to be used in and on houses, the assumed 1 to 3 grams of chips ingested per week would produce an absorbed dose of only 14.5 to 42.8 µg/day.

7.5 EPIDEMIOLOGY

A wide variety of epidemiologic studies have attempted to describe and quantify the health effects associated with lead exposure. The identification of especially susceptible segments of the population has also been a constant objective of these studies. Although both gastrointestinal and respiratory absorption of lead have long been recognized as contributors to body burden, the vast majority of epidemiologic studies have chosen to examine the effects of respiratory, rather than dietary exposures. This apparent emphasis on respiratory exposures relates primarily to the relative ease with which the respiratory exposure level of populations may be estimated (based on suitable environmental monitoring data). Characterizing dietary lead exposures has proven more difficult due to a wider range of individual differences in factors determining lead intake such as absolute caloric intake and composition of diet. Consequently, studies of dietary lead exposures tend to be often laboratory type and generally restrict themselves to intensive, long-term observation of a mere handful of individuals. Several studies of dietary lead exposure are described in Section 7, and alluded to again in Section 8.6, a discussion of the relative importance of dietary vs. respiratory sources to total lead dosage. The remainder of this section, however, is devoted to a discussion of the contribution of airborne lead to blood lead, an indirect indicator of body burden.

Epidemiologic studies have been conducted in three general types of settings based on the characteristic levels of exposure:

(1) Studies of general populations
(2) intermediate level studies in which groups are singled out for study based on some unusual circumstance or mode of exposure. Typically, these studies involve groups chronically exposed to ambient lead concentrations

higher than those of the general population, but substantially lower than industrial exposure levels. Persons residing in close proximity to freeways, working in dense vehicular traffic or living near smelters or other stationary sources of lead emissions are examples of "intermediate" groups for which epidemiologic information is available.

(3) Occupational studies including persons who are exposed to air lead concentrations many times higher than general populations or "intermediate" groups; thus, they represent the high extremes of the dose-response spectrum.

Attempts to establish a dose-response relationship between environmental lead concentrations and adverse health effects and/or the relative contribution of each of the various sources of exposure to body burden have met with only limited success. The underlying difficulty in such an analysis may be traced to the multiplicity of sources of lead exposure and the difficulty in accurately quantifying individual exposure.

7.5.1 Studies of General Population

The risk to general populations from lead in air has become a matter of considerable concern and the subject of many recent studies. As Hammond (1977) has pointed out, studies of the fate of inhaled lead in man using conventional deposition and clearance measurements have not provided much useful information on the contribution of lead in ambient or industrial air to the internal dose at specified air lead concentrations. A more indirect but nonetheless useful approach to the problem proceeds from the assumption that the concentration of lead in the blood is proportional to the combined level of total uptake. Although such a relationship has never been rigorously demonstrated, taken together, the studies discussed in this section place the contribution of air lead to blood lead in the range of about 0.6-2.0 μg/dl in blood per μg lead/m^3 in air.

The earliest large scale study of general populations was the "Three Cities Study" conducted in Los Angeles, Philadelphia, and Cincinnati, Ohio, in the early 1960's (U.S. Department of Health Education and Welfare, 1965). In addition to the general population, several groups more appropriately included in the "intermediate" exposure category (i.e., policemen, traffic controllers, garage employees, etc.) were also investigated as separate study groups. The principal finding of the "Three Cities Study" was that significant differences in blood lead means could not be found between the cities. On the other hand, persons who lived or worked in close vicinity of dense vehicular traffic such as traffic policemen, garage mechanics, and parking lot attendants had higher blood lead levels than others not similarly exposed. These data are shown in Table 7.10.

TABLE 7.10 BLOOD LEAD LEVELS OF SELECTED HUMAN POPULATIONS[a]

Type of Population	Mean Blood Lead, µg/dl	
Population without known occupational exposures:	Male	Females
Remote California mountain	12	(9)
Composite Rural U.S.	16	(10)
Suburban Philadelphia	13	(13)
Composite Urban U.S.	21	(16)
Los Angeles Aircraft workers	19	(17)
Pasadena city employees	19	(12)
Downtown Philadelphia	24	(18)
Population with known occupational exposures:		
Cincinnati policeman	25	
Cincinnati trafficman	30	
Cincinnati automobile test-lane imspectors	31	
Cincinnati garage workers	31	
Boston Sumner-Tunnel employees	30	

[a]Source: U.S. Department of Health, Education, and Welfare (1965).

Goldsmith and Hexter (1967) calculated a logarithmic dose-response regression of blood lead concentrations on air lead levels. The data were mean blood lead levels and the associated average estimated ambient air exposure from the cities included in the "Three Cities Study" (U.S. Dept. of Health, Education, and Welfare, 1965). Under the assumption that the major source of variation in ambient air lead was vehicular traffic, Goldsmith and Hexter calculated particle sizes and consequent retention rates were calculated on the basis of 50-80 percent (by weight) particles < 1.0 µ equivalent diameter. This is believed to be the characteristic particle size associated with automotive emissions. In general, fixed samplers were used to measure ambient levels; although it is recognized that they may not be truly representative of individual exposures, the authors felt they represented reasonable estimates of population averages. Goldsmith and Hexter concluded that a dose-response relationship was suggested when blood lead concentration is plotted against air lead concentration. Data for various groups/individuals are plotted in Figure 7.3 The regression line shown in Figure 7.3 is calculated on the basis of epidemiologic data from the "Three Cities Study" (Goldsmith and Hexter, 1967). In addition, data from four human subjects experimentally exposed to known, high concentrations of lead seoquioxide (Kehoe, 1966) are also plotted along the regression line in order to reinforce the apparent validity of the relationship depicted. The regression line seen in Figure 7.3 is described by the equation:

$$\log_{10} \text{blood lead} = 1.265 + 0.2433 \log_{10} \text{air lead}$$

Thus, the Goldsmith and Hexter regression line may be interpreted to indicate that each microgram of lead in air contributes about 1.3 μg/100ml to blood lead in the range of exposure observed in the "Three Cities Study," (specifically from less than 0.2 μg/m^3 to approximately 8.0 μg/m^3) as shown in Figure 7.3.

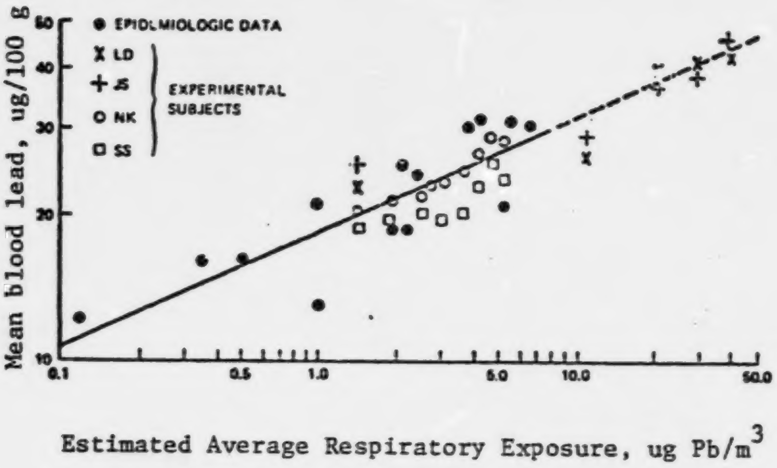


Figure 7.3 Mean blood lead concentration for epidemiologic and experimental respiratory exposures. (Regression from epidemiologic data only.) Redrawn from Goldsmith and Hexter.

Source: National Academy of Sciences, (1972).

The authors concluded that "the close correspondence between the experimental and epidemiologic data (obtained from Kehoe, 1966 and "Three Cities Study" respectively) make it seem likely that this relationship will be valid for a population having a dietary, beverage and cigarette smoking intake similar to that of American males."

Goldsmith and Hexter's proposed dose-response regression has been criticized on several grounds. First, ambient exposures were estimated only and were not necessarily measured at the same times and places at which the populations were exposed. Thus, air and blood levels even within the same exposure group (data points) are not related in any consistent, specific fashion. Second, the validity of calculating a regression line based on lead absorption from atmospheric lead has been challenged on the basis of the variable contribution of lead from other, more important sources of intake such as food and cigarettes (National Academy of Sciences, 1972). Thirdly, the authors do not describe the methods of deriving their regression line nor do they present data on statistical tests of "goodness of fit" of the calculated regression line. Their methods of deriving the estimated "average" exposures levels for occupational groups in policemen, garage workers, and tunnel workers are described only as a weighted average of presumed occupational and ambient exposure which, themselves were estimated. In addition, Goldwater (1972) discusses the matter of confidence limits when this type of statistical analysis is employed. Lines representing confidence limits are hyperbolic and diverge markedly when applied to extrapolations at either end of the regression line; there is little validity to extrapolations beyond the ranges encompassed by actual observations. The NAS report also comments on this phenomenon by stating, "the regression line cannot be applied with confidence to exposure conditions affecting the general population; this also applies to the general population of most large urban centers, inasmuch as average ambient air concentrations even in these centers do not generally exceed 2 $\mu g/m^3$."

In earlier studies such as the "Three Cities Study," multiple uncontrolled variables associated with the various population groups have confused the appraisal of actual integrated exposure and the construction of the correct dose-response relationship. Because blood lead levels are determined by many factors in addition to respiratory absorption of lead in ambient air, the effects of these variables must be either controlled through statistical procedures or made as homogeneous as possible in the initial selection of the study subjects. Unless personal monitoring devices are used, one cannot accurately quantify total integrated exposure over months or years for police officers, garage mechanics, "drivers", "commuters", aircraft workers or other similar groups. Occupational exposures and geographic variability in lead concentrations in various parts of the city are not reflected in air sampling at fixed locations not clearly related to the population in question.

Tepper and Levin (1975) reported the findings of a survey of air and population lead levels in selected American communities, commonly referred to as the "Seven Cities Study". The principal objective of the study was to evaluate the relationship between atmospheric lead levels and the concentrations of lead in blood of persons exposed to the atmospheres in question. Among the cities surveyed were Cincinnati, Philadelphia, and Los Angeles, which had been extensively surveyed in 1961-1962 as part of the "Three Cities Study" previously described. Also included were New York City, Metropolitan Washington, D.C., Greater Chicago, Houston, and Los Alamos, New Mexico. These cities were selected on the basis of population density, industrialization and geographical location to represent a broad spectrum of these variables.

Tepper and Levin (1975) focused particular attention on the definition of populations having a specific, consistent relationship to known air levels of lead. Unlike the Three Cities Study, populations sampled in the present study were women volunteers living within a prescribed distance (within a 1-mile radius) from the reference air sampling station. Moreover, the blood lead and air measurements were made concurrently. In confining the observations to members of one sex, requiring that the subject reside not more than 1 mile from the sampling apparatus, and eliminating as completely as possible extraneous, occupational lead exposures, the authors hoped to create a group which would accurately reflect patterns of respiratory absorption of lead in the ambient atmosphere in a general community setting.

In addition to the groups of women described above, husbands of 100 Los Alamos participants were also included as a separate study group. Estimates of alimentary lead intake were obtained from measurement of the urine and feces of a subsample of 20 volunteers in each region. Eact participant collected all excrement for a 10-day period.

The principal findings of the Seven City Study were as follows:

1) Concerning changes in atmospheric levels of lead during the interval 1961-62 to 1968-69, the reported data demonstrate higher lead levels at most of the study sites during the more recent of the two periods. A careful examination of experimental methodology tends to exclude the possibility that this change is an artifact reflecting changes in technique.

2) The relationship between age and blood lead level was examined by means of regression analysis and was found to be of no general significance.

3) The influence of smoking upon blood lead levels was determined to be clearly significant. The blood lead level of smokers exceeded that of nonsmokers and previous smokers. The relationship held true for males as well as females in the husband-wife pairs in Los Alamos.

4) Since the F-ratio testing the difference in blood lead levels among locations was significant, individual comparisons were conducted between urban and suburban populations in the same metropolitan areas to examine whether or not blood levels reflect degree of urbanization for smokers and nonsmokers. In both smoker and nonsmoker categories, urban groups were consistently higher than nonurban groups. Consequently, it appears that urban blood lead levels are higher than those from related suburban areas.

5) Figure 7.4 presents mean blood lead levels and corresponding mean air lead levels at the sites where both were measured. The association between these mean values was measured by means of the Pearson Product

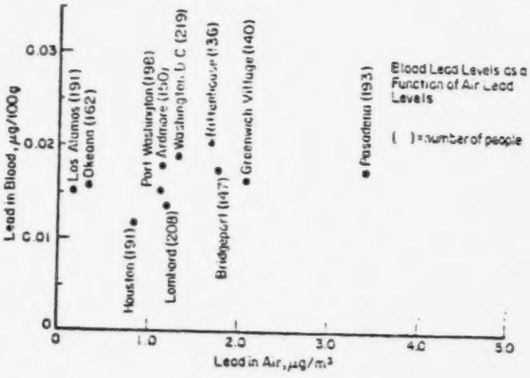


Figure 7.4 Blood lead levels and corresponding mean air lead levels.

Source: Tepper and Levin (1975)

Moment Correlation Coefficient and Kendall's Rank Correlation Coefficient. The values obtained were 0.412 and 0.354, respectively, for all data. Corresponding values for non-smokers only were 0.400 and 0.345. The values were not significant at the 5 percent level. It was therefore concluded that there is no association between average blood and air lead concentrations in the locations studied.

6) The observations that urban levels of blood lead are significantly higher than suburban levels, but that air concentrations of lead are not clearly reflected in blood lead levels generally, suggest that other variables are more important than ambient air lead levels in determining concentrations of lead in the blood. The precise nature of the variables, which evidently differ in the several regions studied, remains undefined. Presumable alimentary lead intake plays a significant role; however this was not clearly evident in the short-term metabolic study. Climatic factors may also be relevant.

7) The analysis of fecal lead levels conducted to appraise alimentary lead intake, did not demonstrate a reciprocal relationship between air and food exposures, i.e. high alimentary intake compensating for respiratory cxposures so as to obscure effects of the latter.

8) The examination of blood lead levels in husband-wife pairs in Los Alamos revealed a significant difference between males and females. The basis for this difference was not established. Probably, it cannot be attributed to occupational factors, for the work of each individual was characterized and conducted under comprehensive and detailed industrial hygiene supervision. Differences in smoking between the sexes could not account for the differences in blood lead, either. The blood lead in males remained higher than in females of the same smoking status.

Since men have generally higher hematocrit levels than women and lead is transported in association with red blood cells, the authors suggested that higher blood lead concentrations in men reflect a higher level of circulating red cells. This hypothesis deserves further investigation. Although both men and women of the studied Los Alamos couples consumed diets presumably similar in composition, the men consumed a generally greater quantity of food. It is therefore also likely that the alimentary exposure of men to lead may be greater than that of women.

In summary, the Seven Cities Study failed to find a significant correlation between air lead and blood lead levels over an air lead range of 0.17-3.39 $\mu g/m^3$. Such a finding should not be unexpected, however, due to the narrow range of ambient conditions examined, the relatively small (20-30 percent) contribution of air to total lead intake (See Section 8.6) at such low exposure levels, and the fact that the air data were obtained from fixed outdoor sampling stations which cannot estimate individual exposure levels precisely.

Azar, et al., (1975) reported on the effects of air lead exposure as measured with personal sampling devices on such indices of lead absorption as blood lead, urine lead, DALA (delta aminolevulinic acid) and ALAD (aminolevulinic acid dehydratase) activity.

The air lead exposure of 30 male subjects in each of 5 locations in the United States was measured with personal air samplers for 24 hours a day for periods ranging from 2-4 weeks. During this period, blood samples were analyzed for lead and ALAD activity analyses and urine samples were measured for lead, DALA excretion, osmolarity and creatinine. Smoking data was obtained by means of a questionnaire. Two types of personal samplers were used: one was an MSA unit equipped with a cyclone device capable of differentiating between respirable and non respirable particles. These were used in the two taxicab studies. The second type was a Casella unit which was more portable, but since it did not have the cyclone device, only total air lead exposure was measurable.

The five sites were selected to represent a wide range of air lead exposures. Taxicab drivers were studied in Philadelphia and Los Angeles. Participants at Starke, Florida, Barksdale, Wisconsin and Los Angeles were employees of Du Pont de Nemours and Company and were thought to be representative of non-occupationally exposed groups.

The average exposure, urine and blood responses were computed for each subject in the study. Statistical analyses were based on these within subject averages. The site averages were subjected to an analysis of variance to determine whether there were any significant differences between average urine and blood responses at the five sites. The relationship between air lead and the blood and urine responses were evaluated using regression techniques. Homogeneity of the slopes were checked by multiple regression techniques and analyses of the data in both natural physical units and logarithmic units (base 10 logarithms of the natural units) were performed. The logarithmic relationship gave the best fit to the date.

Table 7.11 shows air-lead exposure levels which were calculated by combining the work exposure ("on duty") with the home exposure ("off duty") on a time-weighted basis. When a cyclone device was used, the data are referred to as Respirable and when not used, as Total. Exposures ranged from a low of 0.22 $\mu g/m^3$ at Starke to a high of 9.12 $\mu g/m^3$ obtained on a Los Angeles cab driver. Respirable air lead exposures were significantly less than total exposures, indicating that a large portion of "Total" lead is composed of particles >10 microns in diameter which do not penetrate the lung. (These large particles could be swallowed, however.)

Statistical analysis of the data showed that there was no significant correlation between air and blood lead at any of the five sites, indicating either (1) that there is in fact no correlation between air lead and blood lead, or (2) thirty subjects is too few to demonstrate significance, or (3) the range of data is too narrow to show significance, or (4) variables other than air lead (lead ingested from food and drink) are overwhelming any effect which air lead might have on blood lead. Analysis of the composite data (149 subjects) using a multiple regression technique indicated that unspecified variables other than air lead were more important determinants of blood lead.

TABLE 7.11 MEAN AIR LEAD EXPOSURE FOR THE FIVE GROUPS EXAMINED BY AZAR, ET AL (1975)[a]

	Lead Concentration, $\mu g/m^3$ ± 1 S.D.				
	On Duty		Off Duty	Combined Exposure	
Site	Total	Respirable	Total	Total	Respirable
Philadelphia, Pa Cab drivers	5.10 ± 1.11	4.34 ± .86	1.48 ± .23	2.62 ± .42	2.37 ± .34
Starke, Fla.	2.01 ± 2.52	1.39 ± 2.39	0.37 ± .31	0.81 ± .82	0.64 ± 0.78
Barksdale, Wisc.	2.84 ± 5.19	N.D.[b]	0.36 ± 0.14	1.01 ± 1.43	N.D.
Los Angeles, Calif. Cab drivers	9.42 ± .98	7.48 ± .92	4.22 ± 1.28	6.10 ± 1.02	5.37 ± 1.02
Los Angeles, Calif. Office workers	3.05 ± .76	N.D.	3.07 ± .81	3.06 ± .75	N.D.

[a] Source: Azar et al. Reprinted from Lead: Environmental Quality and Safety Supplement, Vol 2, T.B. Griffin, and J. H. Knelson (Eds.) (c) George Thiem Verlag, Stuttgart (1975).

[b] N.D. = Not determined.

Figure 7.5 shows that the data tended to clump into five subgroups by location. The average relationship between air lead exposure and blood lead concentration was obtained by drawing a line with a slope of 0.153 through the average blood lead concentration for 149 subjects. This line is defined by the equation:

$$\log \text{blood Pb} = 1.2257 + 0.153 \log \text{air Pb}$$

The slope of this regression was significantly ($p < 0.01$) different from zero (95% Confidence Limits, C. L. = ± 0.079). Approximately 56 percent of the variance in blood Pb is not explained by air Pb ($R^2 = 0.436$). Figure 7.5 also shows the regression lines obtained when the data from each site are combined and adjusted to a common slope of 0.153. (This procedure is justified since the slopes are homogeneous.) After pooling data from all sites, there was a significant correlation between log air lead level and log blood lead level. The contribution of air lead to blood lead was found to be approximately 1.0 μg of lead per 100 ml of blood per 1 μg per m^3 of air in the range of air lead concentrations studied. This 1:1 estimate is somewhat less than the 1:1.3 ratio estimated from Goldsmith and Hexter's (1967) equation.

The slope of the regression line in Figure 7.5 (0.153) is less than that found by Goldsmith and Hexter (1967) and considerably less than that based on calculations of the Environmental Protection Agency shown as in Figure 7.5 as the "EPA line".

Hammond (1977, personal communication) has applied the mathematical model described by Azar, et al., (1973) to empirical data published by Rabinowitz (1974); Griffin, et al., (1975); Fugas, et al., (1973); Prpic-Majic, et al., (1973); and Williams, et al., (1969) in order to compare actual data to predictions derived from the model.

The formula developed by Azar, et al., (1973; 1975) is as follows:

$$\log \text{PbB} = 1.226 + 0.153 \cdot \log \mu g\ \text{Pb/m}^3$$

where

PbB = blood lead level in μg/dl
Pb/m^3 = air lead level.

The above equation says, in effect, that for this particular population the average PbB at 1 μg Pb/m^3 was:

$$\log \text{PbB} = 1.226 = 16.8\ \mu g/dl$$

since the term (0.153 · log μg Pb/m^3 reduces to zero when air Pb = 1 μg/m^3.

For any other population under study, the constant for PbB at air lead = 1 μg Pb/m^3 will be unique to that population and will depend upon all sources of lead intake. In order to solve for the constant describing log PbB at 1 μg Pb/m^3, baseline PbB at a known small air lead concentration can be used. Taking an example, Coulston, et al., (1972) determined PbB in a group of men known to breathe air lead at 0.2 μg/m^3 throughout the day. Solving for the term b in $a = b + c$.

a = log observed PbB
b = unknown constant
c = 0.153 · log of observed air lead, μg Pb/m^3.

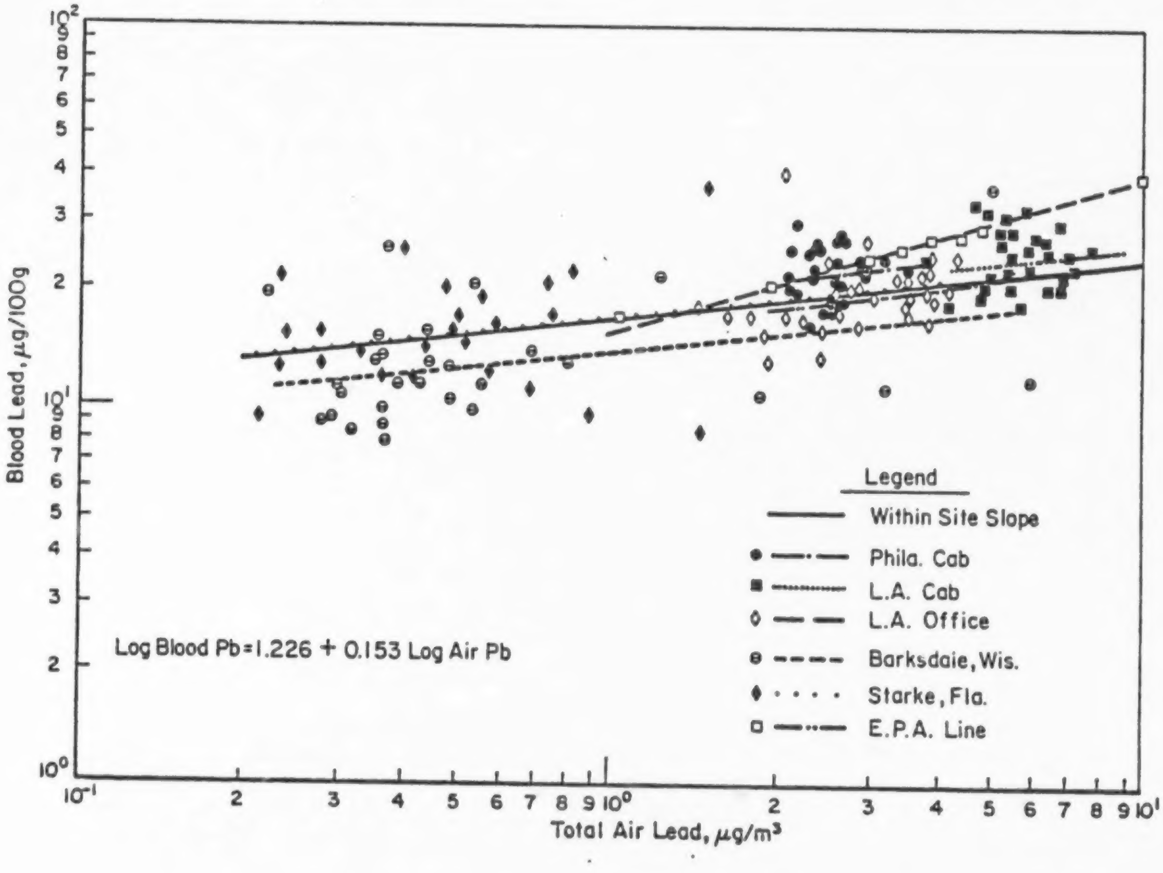


FIGURE 7.5 BLOOD LEAD VERSUS TOTAL AIR LEAD

Source: Azar, et al. Reprinted from Lead: Environmental Quality and Safety Supplement, Vol. 2. T. B. Griffin and J. H. Knelson (Eds.) (c) George Thiem Verlag, Stuttgart, 1975.

Using the Azar model, it is necessary to calculate a new constant for any particular group, log PbB at 1 μg Pb/m^3. In Table 7.12, the baseline data for PbB at low, known air lead is given. Data for log PbB at 1 μg Pb/m^3 are then calculated. This is the constant b described above. Using the calculated constant (log PbB at 1 μg Pb/m^3) the predicted PbB at the high air lead concentration is compared to the actual PbB measured. Finally, the contribution of air lead to PbB as actually observed is given.

Predictions are also provided for PbB values at several air lead concentrations breathed for 40 hours per week against a background PbB of 23.5 with an assumed nonoccupational air lead of 1 μg/m^3.

It is evident from these data that the Azar model for regression of blood lead on air lead predicts fairly accurately the mean blood lead values for groups of workers and human volunteers whose air lead exposures were fairly accurately known. It is also evident that as air lead increases, its impact on blood lead, as designated in the last column of the table, becomes progressively smaller. Thus, Hammond's (1977) data implies that the impact of incremental additions to an already high ambient lead exposure, say over the suggested maximum of 100 μg/m^3 would have only modest impact on internal dose (as reflected by blood lead) relative to equivalent incremental additions at lower air lead levels. Obviously, such a statement should not be construed to suggest that such high industrial levels are safe.

Certain seeming discrepancies occur in Table 7.12. For example, an average air lead exposure of 27 μg/m^3 (Prpic-Majic, 1973; Fugas, 1973) yields a predicted PbB of 46.8 while an average air lead exposure of 72.2 μg/m^3 is predicted (last horizontal row) to yield a somewhat lesser PbB of 45.2. This discrepancy is due to the fact that in the former case PbB at 1 μg/m^3 is 28.3 (antilog 1.4513) while in the latter case it is only 23.5. This only serves to highlight the fact that the impact of air lead on blood lead is very much dependent upon the background of nonair sources of lead.

It may be argued that the Azar model is invalid for industrial situations in which the particle size range and chemical composition of the lead aerosols may be quite different from those encountered in the general ambient air situation prevailing in the Azar study. Nevertheless, the Azar regression equation is quite consistent with the limited experimental data available concerning industrial exposure.

Experimental studies of prolonged exposure to artificially generated lead aerosols have been reported by Griffin, et al., (1975). Lead aerosols were produced by introducing lead-203 tetraethyl lead into gasoline, burning this in an automotive engine, and employing diluted exhaust from the engine as an exposure aerosol for a group of experimental subjects. Thus, the lead aerosol employed in these experiments resembled that of ambient air in particle size but different isotopically. Subjects were exposed for 23 hours a day to lead levels of 3.2 and 10.9 μg/m^3. The experiments were continued for over 4 months to allow time for equilibration of blood lead to the new

TABLE 7.12 THE IMPACT OF AIR LEAD ON BLOOD-LEAD LEVELS; A COMPARISON OF ACTUAL DATA TO PREDICTIONS USING MATHEMATICAL MODEL[a,b]

Baseline			"High" Air Exposure		PbB, $\mu g/dl$			
PbB	at $\mu g/m^3$	log PbB at 1 μg Pb/m^3	μg Pb/m^3	log μg Pb/m^3	Predicted	Actual	μg PbB/ $\mu g/m^3$	Reference
11.2	0.2	1.1561	1.6	0.2041	15.4	12.9	1.2	Rabinowitz, 1974
21.8	0.2	1.4454	3.2	0.5051	33.3	27.6	1.9	Griffin, et al. 1975
20.1	0.2	1.4101	10.9	1.0374	37.1	36.4	1.5	Ditto
22.1	0.2	1.4513	35	1.5441	48.7	44.3	0.64	Fugas, et al., 1973 and Prpic-Majic, et al., 1973
22.1	0.2	1.4513	27	1.4314	46.8	42.9	0.78	Ditto
23.5	1.0	1.3711	51	1.7076	42.9	50.1	0.53	Williams, et al., 1969
23.5	1.0	1.3711	37	1.5682	40.8	39.7	0.45	Ditto
23.5	1.0	1.3711	33	1.5185	40.1	39.5	0.42	"
23.5[c]	1.0	1.3711	24.6	1.3909	38.4	--	--	
23.5[d]	1.0	1.3711	36.5	1.5623	40.7	--	--	
23.5[e]	1.0	1.3711	48.4	1.6848	42.5	--	--	
23.5[f]	1.0	1.3711	72.2	1.8585	45.2	--	--	

[a] Source: Hammond, P. B. (1977, personal communication).

[b] Azar, et al., 1973.

[c] Prediction assuming inhalation of 100 $\mu g/m3$ air lead 40h/wk. + 1 $\mu g/m^3$ for remainder of week (128 h) (avg. = 24.6 $\mu g/m^3$).

[d] Prediction assuming 150 $\mu g/m^3$ 40h/wk. + 1 $\mu g/m^3$ 128/wk. (avg. = 36.5 $\mu g/m^3$).

[e] Prediction assuming 200 $\mu g/m^3$ 40h/wk. + 1 $\mu g/m^3$ 128/wk. (avg. = 48.4 $\mu g/m^3$).

[f] Prediction assuming 300 $\mu g/m^3$ 40h/wk. + 1 $\mu g/m^3$ 128/wk. (avg. = 72.2 $\mu g/m^3$).

exposure levels. The exposures resulted in increases of 2.0 and 1.4 μg/m^3 of blood per 1 μg/m^3 in air. In a similar study, Rabinowitz (1974) removed most of the usual 2.0 μg/m^3 of air lead from the chamber in which one subject was maintained. Following reduction of the air lead, Rabinowitz traced the decline in the blood lead of this subject over the next 40 days. Examination of these data suggest that blood lead appeared to decline approximately 1 μg/dl for each 1 μg/m^3 of lead removed from the air.

Studies have generally reported the regression equation describing the relationship between blood lead and air lead in terms of logarithmic units. Clearly, the contribution of each microgram of lead in air to blood lead (in original units) will change considerably over the range of air lead concentrations examined. Consequently, the ratios above represent the average ratio over a range of air lead concentrations which vary from study to study. These difficulties notwithstanding, there appears to be a fairly good consensus that the contribution of each microgram of air lead to blood lead probably lies in the range from 0.6 to 2.0 μg/dl.

Furthermore, these studies are in close agreement with the data presented earlier by Williams, et al., (1969), Prpic-Majic, et al., (1973), and Fugas, et al., (1973), which estimate a concentration of about 1 μg of lead per 100 ml of blood for each 1 μg/m^3 of air lead exposure.

7.5.2 Intermediate Level Studies

Studies of persons regularly exposed to unusually high ambient lead concentrations (over 10 μg/m^3 for all or part of the day) both from mobile and stationary sources have been much more successful at demonstrating relationships between levels of lead in air and those in blood. Actually, it is only under these circumstances that respiratory intake of lead accounts for a substantial percentage of total absorption. It should be mentioned that few of the persons in the intermediate category are exposed to sustained levels of over 10 μg/m^3 for more than a few hours a day.

Chronic exposure to intermediate levels of lead from mobile sources may occur either as a result of residing in close proximity to high traffic density (e.g., freeways) or from occupations which involve exposure to above average amounts of automotive exhaust either indoors or outdoors. Policemen, taxi drivers, garage attendants and mechanics are the most common examples. Populations residing near stationary sources of lead emissions such as primary and secondary smelters and battery plants have also been studied. The effects of exposure to higher than average amounts of lead among the Wenatchee orchardists and their families has also been reported.

7.5.2.1 Mobile Emission Sources--

Thomas, et al., (1967) conducted a study to determine whether persons living near freeways in Los Angeles County have increased levels of lead in blood. Fifty adults who had resided for at least 3 years within 76.2 meters (250 feet) of a freeway were compared with 50 who had resided for a like period near the ocean or at least 1.6 kilometers (1 mile) from a freeway. Average blood-lead levels were substantially higher in the population sample living near the freeway. However, these blood-lead levels were similar to other Los Angeles populations and lower than those reported for some other urban populations. The observed difference between the two population samples is consistent with the existence of coastal-inland atmospheric lead and blood-lead gradients within the Los Angeles Basin.

The effect of distance of residence from highways upon blood-lead levels among black females has been investigated by Daines, et al., (1972) (see Table 8.5). Average annual air-lead levels on the front porches of homes located 3.7, 38.1, and 121.9 meters away from a highway were 4.60, 2.41, and 2.24 micrograms per cubic meter, respectively. There was no significant difference between the outside air-lead concentrations at 38 and 122 meters, but both were different from the air-lead concentration at 3.7 meters from the highway. This rapid nonlinear decrease in airborne lead with distance from the source has been observed for mobile as well as stationary lead sources. Concentrations of lead in dustfall also decreased rapidly with increasing distance from the roadway. Indoor air-lead levels measured inside the front room of houses at the three sampling points also reflected this nonlinear decrease, being 2.3, 1.50, and 1.57 micrograms per cubic meter as the distances increased.

Average blood-lead leavels in the study subjects wer 23.1, 17.4, and 17.6 μg/100 m^3 of blood at 3.7, 38.1, and 121.9 meters, respectively. Just as the air lead levels, the average blood-lead levels at the two more distant sampling sites differed significantly from the average at 3.7 meters, but did not differ significantly from one another. Among the homes closest to the highway (3.7 meters), air-lead and blood-lead levels were significantly lower for subjects whose homes were air-conditioned than for similar subjects in homes without air-conditioning.

. Despite the fact that the air lead levels measured at the two locations differed from one another statistically, this difference would not necessarily be reflected in higher blood lead levels at the nearer site. Under the assumption that each microgram of lead per cubic meter of air contributes 0.6 to 2.0 μg of lead per 100 ml of blood, one would not expect that the difference in ambient lead exposure between the two sites (0.22 μg/m^3) to result in differences in blood lead of more than about 0.4 μg lead/dl.

Groups exposed to above-average amounts of automotive exhaust outdoors, such as policemen, may or may not have lead levels above those of comparable groups not so exposed according to a U.S. Environmental Protection Agency (1977) report. Differences, when present, are small (10 to 20 percent), but ranges are sometimes above normal limits. However, groups similarly exposed indoors (U.S. Dept. of Health, Education and Welfare, 1965; Tola, et al., 1972; Gothe, et al., 1973) such as parking garage employees, consistently have 20 to 40 percent higher levels than ordinary groups, with greater numbers of individuals having blood leads above the normal range (blood lead of 40 μg/dl or higher). Groups exposed to lead at controlled stationary sources of lead production have blood-lead levels similar to those of parking garage employees or those of individuals exposed in automotive repair facilities. Groups exposed in automotive repair facilities (U.S. Dept. of Health, Education and Welfare, 1965; Tola et al., 1972) consistently have higher levels (20 to 100 percent higher) because exposure to solder, batteries, etc., is added to exposure to automotive exhaust. The range of levels is increased, and many are above 40 μg/dl as demonstrated in the Three Cities Study.

Lawther, et al., (1972) studied airborne inorganic lead and its uptake by inhalation using three different types of data: (1) measurements of airborne concentrations of inorganic lead compounds taken in a busy London street and at a control site away from traffic, (2) measurements of the blood-lead concentrations in London taxi drivers using day workers as the test group and night workers as the controls; and (3) visual appearance of particles collected from the general atmosphere of London, from a busy London street and from the exhaust gases of petroleum and diesel engines.

Blood-lead and carboxyhemoglobin levels were studied among London taxi drivers on both the day and the night shift. Significant differences were found in blood carboxyhemoglobin, but not in blood-lead levels, when comparing smoking and non-smoking day-shift drivers to similar night-shift drivers. Lawther, et al., (1972) concluded that the day drivers had a greater exposure to motor car exhaust than the night-shift drivers because they had significantly higher blood carboxyhemoglobin levels and therefore "it would seem that little of the lead found in their blood is attributable to the lead they inhale while driving in London streets". Unfortunately, the report does not provide any evidence of distinctively different exposures of day and night drivers in terms of ambient lead concentration. Also, the fact that differences in carboxyhemoglobin (COHb) levels were found in day and night drivers does not really constitute valid evidence of differences in lead exposure. Because the biological half-time of carboxyhemoglobin in blood is 3 or 4 hours one might expect not only rapid equilibration of blood COHb with atmospheric CO but also a diurnal variation in COHb levels among the cab drivers. On the other hand, Kehoe demonstrated that the half-time of lead may be as long as several weeks (Kehoe, 1960) and similar diurnal patterns of blood lead levels would not be expected. Consequently, the COHb cannot be used as a valid indicator of lead exposure from automotive emissions. Furthermore, information was not obtained regarding the exposure of the drivers when they were off duty (U.S. Environmental Protection Agency, 1972). Thus, the relative contribution of automotive exhaust versus other sources of lead remains unresolved.

Iwata, et al., (1971) described the effects of lead in auto exhaust on traffic controllers. The study measured lead in areas representative of the controller's work environment, along with medical examinations for signs of lead toxicity. Lead concentrations in air ranged from 3.3. to 916 $\mu g/m^3$. Medical examinations were carried out on 17 traffic controllers (Group I) and 14 indoor policemen (Group II). Subjective symptoms included headache and nettle rash, sore throat, nausea, constipation, lumbago, and dizziness. More complaints were found in Group I. No difference was found between the two groups in terms of whole blood specific gravity, amount of blood pigment, and punctate, basophilic erythrocyte level. The average level of lead in blood ranged from 12.3 to 24.5 μg/dl in Group I and 10.1 to 17.3 μg/dl in Group II, indicating slightly higher levels in Group I, however, these differences were not large enough to be statistically significant.

Landrigan, et al., (1975c) discovered three cases of mild lead poisoning among instructors at an indoor pistol range. These cases were characterized by blood-lead levels greater than 100 μg/dl of blood, free erythrocyte protoporphyrin levels greater than 450 μg/dl of red blood cells, abdominal pain, and, in one instance, by slowing of motor and sensory nerve conduction velocity. Fragmentation of bullets during firing, explosive vaporization of primer, and aerosolization of lead suboxide particles during bullett molding appear to have been the major sources of airborne lead.

7.5.2.2 Populations Impacted by Stationary Sources--

Stationary sources of lead emissions can be divided into three categories:

1) Primary smelters where the lead bearing ore is processed. Because lead ore generally will contain other metals (such as zinc, cadmium, bismuth, and arsenic), effects observed in exposure studies may not always be caused by lead alone. Consequently, establishing a dose response relationship for health effects in such studies is difficult due to the confounding effects of other potentially toxic emissions.

2) Secondary lead smelters and lead processing plants.

3) Miscellaneous sources, consisting of those industrial processes where the main objective is not to obtain or process lead. Examples include battery manufacture, painting or torch cutting surfaces coated with leaded paint, cable stripping, and other operations in which lead is incidentally released into the environment.

7.5.2.2.1 Primary smelters--A recent Environmental Protection Agency report has summarized very well the studies which have examined the effects of lead exposure on persons residing near stationary lead emitting sources (U.S. Environmental Protection Agency, 1977). Extensive investigations of the effects of lead exposure on people living near primary smelters have been performed in El Paso, Texas, Helena Valley, Montana, and Kellogg, Idaho. Several of these studies are briefly summarized below.

The smelter at El Paso, Texas, has been operating since the late 1800's and produces not only lead but also copper and zinc (Landrigan et al., 1975a; Texas Morbidity and Mortality Weekly Report, 1973). In addition to these metals, cadmium and arsenic are known to be emitted. The estimated lead emissions were 292 metric tons in 1968; 511 metric tons in 1970, and 312 tons in 1971. These figures, however, are only for stack emissions; it is not known how much lead may have been emitted from fugitive emissions via ventilation, windows, etc. An epidemiologic study focused on the so-called Smeltertown Section, a residential area less than 182.88 meters (200 yards) west of the smelter, as well as some nearby communities farther downwind that were also affected by the smelter emissions. The concentrations of lead in air are presented in detail by U.S. Environmental Protection Agency (1977) and in the published reports referred to above. Daily lead concentrations in Smeltertown in 1972 ranged from 0.49 to 75.0 $\mu g/m^3$. For 86 sampling days in 1972, the average level was 6.6 $\mu g/m^3$. Air lead levels fell off rapidly with increasing distance from the smelter, declining to background levels at 4 to 5 km from the smelter (see Figure 8.2). High concentrations of lead were found in the soil, being greatest in the area nearest the smelter (geometric mean 1791 ppm lead). High concentrations of lead in house dust were also found, ranging from 400 to 58,800 ppm.

Determinations of lead in blood revealed that 53 percent of the children aged 1-9 residing within a distance of 1 mile or less from the smelter had blood lead levels > 40 μg/dl, the level considered by many investigators to be indicative of undue lead absorption. Of children residing between 1 and approximately 4 miles (6.6 km) from the smelter, 18 percent had blood lead levels above 40μg/dl. Beyond that distance and among older children (over 9) levels in blood were lower. Airborne lead levels of 8-10 $\mu g/m^3$ annual mean were recorded in the area within 1 mile of the smelter. Household sampling revealed that children within this 1 mile area with blood leads ≥ 40 μg/dl were exposed to as much as 3.1 times as much lead in dust (6447 vs. 2067 ppm) as the children with lower blood values. Paint, water, food and pottery were also examined as potential sources of lead, however the authors stated that they believed particulate lead in air and dust accounted for most of the undue lead absorption in El Paso children (Landrigan, et al., 1975a).

A smelting complex in East Helena, Montana has also been investigated as a source of airborne air pollution. Zinc, cadmium, and arsenic are known to be emitted also, however the quantity of lead emissions is not known (U.S. Environmental Protection Agency, 1977) Air concentrations of lead were measured in 1969, and in East Helena the average concentration at several stations varied from 0.4 to 4 $\mu g/m^3$; the maximum 24-hour value was found to be 15 $\mu g/m^3$. In the city of Helena, the average concentration was 0.1 $\mu g/m^3$. Lead in soil was found to be 4000, 600, and 100 ppm at distances of 1.6, 3.2, and 6.4 km (1, 2, and 4 miles), respectively, from the smelting complex; whereas, in uncontaminated soil near the Helena Valley it was 16 ppm. Deposited lead (dustfall) was found to vary from 3 to 108 mg/m^2/month in East Helena; whereas, in Helena it varied from 1 to 7 mg/m^2/month.

Studies on humans were limited to children; lead in hair and blood were found to be higher in East Helena than in Helena. The respective averages were 15.6 and 11.6 μg/dl in blood and about 40 and 13 ppm lead in hair (Hammer, et al., 1972). Both blood lead and hair lead are suggestive of greater exposures nearer the smelter. The blood values reported indicate that lead absorption, although evidently elevated in residents near the smelter was still essentially within normal limits. No other adverse health effects have been noted among these children.

The U.S. Environmental Protection Agency (1977) reported preliminary data on air lead in the vicinity of a lead-zinc smelter at Kellogg, Idaho which showed that during 6 months in 1971 the average concentration of lead in air was between 6 and 8 $\mu g/m^3$. Children from this area had a mean blood level of 20.9 μg/dl and a mean hair-lead level of about 100 ppm. Based on lead in air determinations, the exposure seems to be about twice as high as in East Helena, which is supported by the finding that lead in hair was twice as high. This exposure resulted in about 5 μg/dl higher lead values in blood.

In September 1973, a fire destroyed the main fume filtration system at the smelter, causing increased lead emissions. Records showed that emissions of lead to the atmosphere averaged 8.3 metric tons per month from 1955-1964, 11.7 metric tons from 1965-Sept. 1973 and 35.3 metric tons from October 1973 to Sept. 1974 (Landrigan, et al., 1976). Following the hospitalization of two children with symptoms of lead poisoning, the Center for Disease Control joined with the State of Idaho in a comprehensive study of children in the area (Landrigan, et al., 1976; Yankel, et al., 1977).

In September, 1974, blood-lead levels $\geq$ 40 µg/dl were found in 385 (41.9 percent) of 919 children less than 10 years old who were examined. About 99 percent of the 172 children living within 1.6 km of the smelter had blood-lead values $\geq$ 40 µg/dl. The mean blood concentration declined with distance from the point of emission. Blood-lead levels were consistently higher in children 1 to 4 years old than in those 5 to 9 years old. In addition, higher levels in children were associated with reported active ingestion of lead-containing material (pica), with lower socioeconomic status, and with parental employment at the smelter or at a lead mine. A significant negative relationship between blood-lead level and hematocrit value was found. Seven of 41 children (17 percent) with blood-lead levels $\geq$ 80 µg/dl were diagnosed by the investigators as being anemic on the basis of hematocrit less than 33 percent, whereas only 16 of 1006 children (1.6 percent) with blood-lead levels < 80 µg/dl were so diagnosed (U.S. Environmental Protection Agency, 1977a).

Subsequently, Yankel, et al., (1977) published additional information concerning the 1974 study as well as the results of a follow-up study conducted in 1975. The follow-up was undertaken to determine the effectiveness of control measures initiated after the 1974 study.

Between August 1974 and August 1975, the mean annual air-lead levels decreased at all stations monitored. In order of increasing distance from the smelter, the relative concentrations in the two years were 18.0 to 10.3 µg/m^3, 14.0 to 8.5 µg/m^3, 6.7 to 4.9 µg/m^3, and, finally, 3.1 to 2.5 µg/m^3 at 10 to 24 km. Similar reductions were noted in the housedust-lead concentrations (U.S. Environmental Protection Agency, 1977a).

The report of Yankel, et al., showed that there were reductions in environmental lead contamination between 1974 and 1975, and that the correlations between blood-lead levels and environmental or demographic factors were consistant from one year to the next. Five factors significantly influenced the probability of a child's developing an excessive blood-lead level:

1) Concentrations of lead in ambient air (µg/m^3)

2) Concentration of lead in soil (ppm).

3) Age (years).

4) Cleanliness rating of the home (subjective evaluation coded 0, 1 and 2, with 2 signifying "dirtiest").

5) General classification of the parents' occupation.

These investigators developed a matematical model based on the 1974 data that included each of the five factors that had been shown to be correlated with increased blood-lead levels. The model, shown below, can be used to estimate the effect of variations in the environmental factors on mean blood-lead levels in children:

$$\ln (\text{Pb-B}) = 3.1 + 0.041 (\text{Air}) + 2.1 \times 10^{-5} (\text{Soil}) + 0.087 (\text{Dust}) + 0.018 (\text{Age}) + 0.024 (\text{Occupation}).$$

Although the strongest correlation found was between blood lead level and air-lead level, the authors concluded that it was unlikely that inhalation of contaminated air alone could explain the elevated blood-lead levels observed. In another report, Von Lindern and Yankel reasoned that even though air lead was the principal source, a major route of exposure was the ingestion of lead in soil and dust. They proposed that to protect the health of the children in this area the regulation of environmental standards must take into account all of these routes of exposure (U.S. Environmental Protection Agency, 1977a).

It is clear from the preceding discussion that emissions from primary smelters may cause elevated blood levels and other signs of increased lead burdens in populations living near the emission sources. In El Paso and Kellogg, the high concentrations of lead in blood of children in these areas are comparable at or above levels associated with increases in ALA excretion. Elevated lead levels in blood and urine and increases in ALA-U are, of course, results of lead exposure. As pointed out earlier, primary lead smelters will emit other metals, and nonspecific effects may well be caused by these or other contaminants (U.S. Environmental Protection Agency, 1977). Efforts should be made to separate effects of such contaminants from lead effects.

A recent EPA report (U.S. Environmental Protection Agency, 1976) investigated mortality patterns in counties containing primary smelters in comparison with contiguous, non-smelter counties to determine whether smelter emissions produced any noticeable health effects in terms of disease-specific mortality.

Several findings were consistent among all, or most of the sites examined. A majority of smelter counties showed an excess of deaths from five categories of deaths:

1. All causes combined;
2. Cancer of the liver and biliary passages;
3. Cancer of the trachea, lung and bronchus;
4. Cancer of the kidney; and
5. Cancer of the bladder.

Excesses from other non-cancer causes of death were frequently found in one or two of the smelter counties. These excesses were predominantly from respiratory diseases such as bronchitis, emphysema, asthma and pneumonia. Excess deaths from arteriosclerosis and small vessel disease were also seen in some smelter counties, however this pattern was not consistent.

These findings appear to conflict with the results of other studies. Traditionally, lead has not been thought to be a carcinogen in man, although it has been shown to induce cancer in rodents. Similarly, cancer has not been shown to be excessive in several studies of cohorts of lead workers. The study cited above suffers from several methodological weaknessess which cast doubt on the validity of the findings. First, the failure to eliminate, control for, or at least quantify the levels of other known carcinogens present in smelter emissions suggests that even though there may be a genuine excess of cancer in the smelter counties, the causal connection between lead exposure and cancer has not been established convincingly. Second, in comparative studies of inter-community variation in health status, it is essential that demographic factors independently associated with mortality risk (i.e. population density, age, race, and sex distributions, socioeconomic and educational levels) be explicitly controlled in the analysis, lest effects attributable to differing population compositions in the areas being compared be erroneously ascribed to effects of exposure. Adjustments for these factors in this study were remarkably inadequate. Consequently, findings from this particular study should be viewed with skepticism, however, this means of approach offers some possibilities if an adequate study design could be developed.

7.5.2.2.2 Secondary smelters and lead processing plants--A good example of exposure from secondary smelters has recently been given by Nordman, et al., (1973). A random sample of 621 individuals 16 years old or older in an area near a secondary smelter in Finland was selected; 334 individuals responded, of whom 41 were rejected for various reasons. Lead and ALAD in blood were determined in the remaining 293 individuals. The results were related to distance from the source and to dustfall levels of lead, which had been determined at 80 stations during a 1-month period. Significant negative correlations were found between distance from source and lead or ALAD in blood. This was especially pronounced in a group of women who spend most of their time at home. On a group basis, lead in blood was also found to be correlated to dustfall. However, blood-lead levels were generally below 30 μg/dl, and only in a group of males living in a zone where the dustfall was about 100 mg/m^2/month was the mean blood level 30 μg/dl. Dustfall in this study was about the same as that reported from East Helena.

Exposure from both a primary and secondary smelter in the inner city area of Omaha, Nebraska, has been reported (McIntyre and Angle, 1972; Angle and McIntyre, 1974). In the report, the authors did not take into account the city traffic, but in a more recent study reported by U.S. Environmental Protection Agency (1976) these authors concluded that elevated blood levels of lead could be related to proximity to a battery plant, traffic intensity, and substandard housing. These reports illustrate some of the difficulties involved in studying exposures from point sources in cities.

Martin (1974) reported an epidemiological survey of lead works of various types to determine possible adverse health effects on mothers and children living in the area. At a point approximately 100 meters from the factory chimney, average atmospheric lead concentrations of 3.0 μg/m^3 with occasional 24-hour concentrations ranging up to a maximum of 28 μg/m^3 were found. An extensive series of measurements of lead deposits was made in the vicinity and monthly figures of up to 392 μg/m^2 were found; environmental dusts contained concentrations of up to 5 percent, and soil concentrations ranged from 0.15 to 4.9 percent. Initial results indicated that of 39 children under the age of 5 years living within 400 meters of the factory, 41 percent had blood levels of above 40 μg/dl whereas of 80 children living at distances of 400 to 500 meters from the factory, only 13.7 percent had blood-lead values above this level. Similarly, of 252 children attending local schools, 44 were found with blood levels above 40 μg/dl, 6 of these being above 60 μg/dl. Of the 26 mothers living within the 400 meter range, 3 had blood-lead levels over 40 μg/dl, whereas of 53 living in the 400-to 500-meter range in none did the blood lead exceed this level. Of the 4 children under shcool age with the highest blood leads, 3 (with levels of 75, 74, and 65 μg/dl) were living close to the works and 2 of these had fathers working there. In the third, the child's only known exposure to abnormal quantities of lead was the result of his proximity to the works. In the fourth, who lived in the 400- to 500-meter range, there was evidence of pica. Where raised blood levels were found, investigations were carried out in the home to exclude as far as possible sources of raised lead intake other than those derived from the factory. Apart from the occasional case of pica, no abnormal source was established and the principal cause of the raised levels was presumably either emissions to the atmosphere from the factory, windborne dusts, or dusts from vehicles entering and leaving the works, or lead taken home on the person or clothing of a parent working in the factory.

Roberts, et al., (1974) report on the dispersal of lead and on the health effects of lead contamination around two secondary smelters located in different parts of Toronto. Each smokestack is about 100 meters south of a residential area and 100 to 200 meters north of an elevated expressway carrying 50,000 to 150,000 cars per day. Lead emissions from the two smelters were estimated at 15,000 to 30,000 kilograms per year. The high rate of lead fallout around the secondary lead smelters was attributed to episodal large-particulate emissions from low-level fugitive sources rather than from stack fumes. The lead content of dustfall, and consequently of soil, vegetation, and outdoor dust, decreased exponentially with distance from the two smelters. Monthly geometric means of the lead concentrations in suspended particulates close to the smelters ranged from 1 to 5.3 μg/m^3 in air or approximately twice that of many urban sites.

Blood samples for lead analyses were collected from 286 people close to the first smelter, 1425 people close to the second smelter, an urban control group of 1231 individuals of similar socioeconomic status. To determine the major source of increased lead absorption, blood and hair samples were obtained during the same period as the above from 16 families living within 15 meters of the first smelter and 8 urban control families living in houses of similar age and upkeep.

Between 13 and 30 percent of the children living in the contaminated areas had absorbed excessive amounts of lead (as evidenced by blood lead of over 40 μg/dl and more than 100 micrograms of lead per gram of hair) compared with less than 1 percent among the control group. A relationshiop between blood and hair lead was established which indicated that the absorption was fairly constant for most children examined. Consequently it appeared that the ingestion of contaminated dirt and dusts rather than "paint pica" was the major route of lead intake. Metabolic changes (i.e. increased urinary excretion of ALA and coproporphyrins) were found in most of the 21 children selected from those with excessive lead absorption; 10 to 15 percent of this group showed nonspecific neurological dysfunctions and/or reduction in peripheral nerve conduction velocity.

7.5.2.2.3 Miscellaneous sources--There have been very few epidemiologic studies of groups exposed to lead in which a large number of health parameters were measured and the results in different exposure groups compared. One of the earliest of such studies concerned the orchardists in the Wenatchee area of Washington state who were exposed to lead arsenate as reported by Neal, et al., (1941) and followed up in a later study by Nelson, et al., (1973).

The lead concentrations observed were not as high as those sometimes seen in other industrial exposures, but that makes them particularly important because the urine-lead concentrations range from slightly higher than commonly found in urban communities to the same as in moderate lead exposures in industry.

Among the factors studied in assessing the health of the orchardists were weight, blood pressure, diseases of the cardiovascular system, skin disorders, eye irritation, chronic nervous diseases, blood dyscrasias, kidney disease, pulmonary tuberculosis, visual acuity, syphilis, neoplastic disease, and fertility. Each parameter was studied to find out whether it had been modified by the lead arsenate exposure. Insofar as comparative data for other populations were available, no evidence was found that any of these factors was altered by the exposure. Special attention was given to the medical examination of children because in the Wenatchee area, where orchards surrounded the communities or the houses in which they lived, there were unusual opportunities for children to be exposed to lead arsenate insecticide sprays and spray residues on branches, leaves, and grass, in addition to residues ingested on apples. In only one respect did these children differ from children in other districts: their urinary lead and arsenic concentrations were nearly twice as high as those of a group of 18 children measured at the same time in Washington, D.C. (who had a mean urine lead content of 0.025 mg/l; standard deviation, 0.0128).

Serious questions might be raised concerning the relative importance of the lead compared with the arsenical portion of the lead arsenate molecule in the overall toxicity of the compound since any toxic action observed might be attributable to arsenic. The National Academy of Sciences (1972) reported that findings from animal studies indicate that the greater toxicity of the lead arsenate molecule is attributable to the lead radical rather than to the arsenic; no synergistic action of lead and arsenic was reported. Based on its review of the Wenatchee orchardists study, the same authors concluded that there was no indication of adverse effects of lead arsenate exposure on the health of the Wenatchee children (National Academy of Sciences, 1972).

7.5.3 Studies of Occupational Groups

Occupational lead exposures represent the high end of the dose-response spectrum. It is among workers who smelt, refine, and manufacture lead-containing or lead-painted products that the highest and most prolonged lead exposures are seen. Consequently, the study of occupational groups provides valuable data on maximal human health effects in terms of both high ambient concentration and chronicity of exposure. At the outset it should be noted that the clinical pattern of occupational lead poisoning has changed during the 20th century in that the incidence and severity of poisoning has decreased substantially in recent years. Whereas acute and occasionally fatal lead poisoning was seen early in the 20th century, today's cases of lead intoxication are frequently diagnosed at an earlier, asymptomatic stage on the basis of abnormalities of biochemical, neurological, or renal function. Nevertheless, excessive lead absorption and its attendant health effects remain a serious and prevalent occupational hazard in the lead industries today. The findings of several recent surveys reported in this section indicate that present standards and work practices of the lead industry do not adequately protect lead workers from adverse health effects.

7.5.3.1 Threshold Levels for Clinical and Subclinical Effects--

It is perhaps useful in this discussion to work downward from the worst effects which are universally regarded as adverse to health to those which are of uncertain health significance. In this regard, it is customary to distinguish between clinical and subclinical effects. For the purpose of this review, clinical effects are those which a subject can perceive and which a physician would normally seek to correct (i.e. biological changes known to be indicative of disease). Subclinical effects are changes of a more subtle nature and which, under normal circumstances do not result in a perceptible decrement in bodily function. Such changes could possibly reduce an individual's capability to cope with a coexistent body stress (Hammond, 1978). Subclinical effects typically occur in asymptomatic individuals and are usually detectable only by sophisticated and generally non-routine biological tests.

7.5.3.1.1 Dose-response relationships for clinical effects--Lead intoxication, in its severest forms, can cause permanent damage to the body or death. Observed clinical effects include damage to: (1) the central nervous system, including the brain, (i.e. acute and chronic encephalopathy), (2) the peripheral nervous system, (3) the kidneys and (4) hematopoietic system (i.e. anemia). Symptoms which may vary in severity include colic, abdominal pain, loss of appetite, constipation, excessive tiredness and weakness, nervous irritability, fine tremors, and weakness in the extensor muscles of the hands and feet (U.S. Department of Labor, 1975).

A number of studies have sought to relate clinical symptoms and effects caused by lead exposure to workers' blood-lead levels (National Academy of Sciences, 1972; Williams, 1975). Kehoe, (1961 a-d) based on observations of occupationally-exposed men who were otherwise healthy, reports that clear-cut symptoms of acute lead intoxication are associated with a blood-lead level over 80 μg/dl of whole blood. Moreover, he observed that as concentrations of blood lead rise above 80 μg/dl, the likelihood of lead intoxication increases. Although such concentrations do not necessarily result in lead intoxication in all individuals, the probability of lead intoxication increases dramatically at blood lead levels above 80 μg/dl as shown in Figure 7.6 (Williams, 1975). In addition, a number of studies have observed symptoms and effects caused by exposure to lead at blood-levels below 80 μg/dl (National Academy of Sciences, 1972; U.S. Department of Labor, 1975).

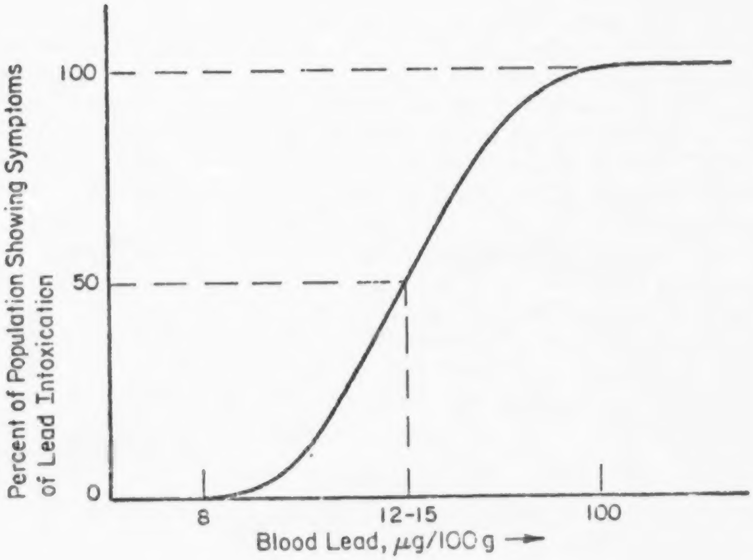


Figure 7.6 Hypothetical distribution curves of blood lead concentrations of a population exposed first to 200 mg Pb/m^3 then to 500 mg/m^3

Source: Williams, (1975). Reprinted, with permission from J. Occupational Medicine. (c) Am. Occup. Med. Assoc. (1975).

The Occupational Safety and Health Administration has concluded, "while 80 μg/dl is a useful lower range for observed clear-cut clinical symptoms, we do not regard it as a sharp delineation above which clear-cut symptoms occur in all workers and below which clear-cut symptoms do not occur. Further, workers with blood-lead levels above 80 μg/dl without clear-cut symptoms may have milder symptoms caused by lead exposure. It should be noted that in evaluating studies which seek to relate blood lead levels to symptoms of lead exposure, it is rarely possible in clinical situations to determine the amount of lead absorbed before the onset of symptoms of lead intoxication."

Gibson, et al., (1968) examined a series of 100 workers from different lead industries. Ninety one of these individuals were working and 9 were hospitalized for lead poisoning at the time of the study. The objectives of this investigation were to define more clearly the clinical and biochemical criteria of lead poisoning in three stages: (1) a presymptomatic stage of lead exposure (37 men); (2) a stage involving mild symptoms or mild anemia (45 men); and (3) frank lead poisoning with severe symptoms and signs (18 men). Urinary lead values were similar for all three groups; blood-lead tests were less sensitive in discriminating symptomatic cases than hemoglobin and urinary coproporphyrin, and delta-aminolevulinic acid (ALA) estimations. Although the latter three parameters correlated well with each other, they were uncorrelated with urinary and blood-lead levels. Porphobilinogen (PBG) levels became raised with symptoms of lead poisoning. A hemoglobin of 13 grams per 100 milliliters or less was considered a cautionary sign. Urinary coproporphyrin above 80 micrograms per 100 milligrams creatinine, ALA above 2.0 milligrams per 100 milligrams creatinine, and PBG above 0.15 milligram per 100 milligrams of creatinine were always associated with symptoms and were therefore considered to be the upper safety limits. Blood-lead values of 60 micrograms per 100 grams of blood were proposed as the upper acceptable limit.

Among the occupational groups represented in this survey, scrap metal burning, battery manufacturing, and shipbreaking constituted the gravest lead hazards, whereas wire manufacturing constituted the least. Workers in the most modern factory, a car-body pressing plant, had urinary coproporphyrin and ALA estimations just below the danger level for these parameters despite apparently efficient protective measures. This observation underscores the importance of medical supervision of lead workers.

Epidemiologic studies conducted between 1975 and 1976 in five different lead facilities across the United States have shown unacceptably high blood-lead levels and symptoms of lead poisoning in every plant studied (Center for Disease Control, 1977). The sites investigated included four secondary smelters and one lead chemicals plant (Eagle-Picher Industries, Joplin, Missouri), which produces such lead compounds as lead oxides, lead peroxide, lead sulfate, and lead silicate.

Information concerning the plants included in the investigation is given in Table 7.13. Hematologic, neurologic and renal damage due to lead were consistently reported. Inappropriate medical management (misuse of chelating drugs) and poisoning of workers' children from home contamination with lead dust were also observed in one or more of the plants.

Initial investigations at the Memphis, Tennessee, secondary lead smelter were prompted by reports of excessive blood-lead levels and clinical

TABLE 7.13 DESCRIPTIONS OF LEAD PLANTS INVESTIGATED, 1975-1976[a]

Date of Study	Location	Type of Plant	Date Plant Began Lead Processing	Pounds of Lead Processed per Month
November 1975	Memphis, Tenn.	Secondary smelter	1948	2.3 million
March 1976	Joplin, Missouri	Lead chemicals plant	<1930	--
May 1976	Eagan, Minnesota	Secondary smelter	1948	1.5 million
January 1976	Salt Lake City, Utah	Secondary smelter	1975	30,000-250,000
March 1976	Atlanta, Georgia	Secondary smelter	--	--

[a] Source: Center for Disease Control, 1977.

lead poisoning at this plant. Ninety-two percent of current employees and one former employee were examined, revealing blood-lead levels up to 184 μg/dl with 67 percent above the currently proposed occupational standard of 60 μg/dl. Table 7.14 summarizes the distribution of blood-lead levels at the Memphis facility as well as from the other four sites. The highest levels were seen in production area workers, who exhibited incidence rates of classical signs of lead poisoning as follows:

Abdominal pain (17 percent)
Gastrointestinal dysfunction (22 percent)
Joint pain (28 percent)
Neuromuscular symptoms (27 percent)
Anorexia (23 percent)
Lead neuropathy (8.5 percent) weakness of wrist extensor muscles
Anemia (13 percent) defined by hemoglobin $\leq$ 14 μg/dl

Three workers were anemic, and neurological symptoms including wrist weakness, ankle drop and tremors were noted in a few workers. Symptoms compatible with lead poisoning were noted in 38 percent of the 53 workers examined.

The most noteworthy finding at the Memphis plant was the high incidence of renal disease; 32 percent of the workers had elevated blood urea nitrogen (BUN $\geq$ 20 mg/dl). Further studies revealed significant impairment of renal tubular and glomerular function in 7 workers, a finding which may represent early stages of lead nephropathy.

Investigations at the secondary lead smelter at Salt Lake City, Utah, revealed unequivocal lead poisoning in 52 percent of those examined; two hospitalized workers had blood lead levels over 250 μg/dl. In addition, 36 percent of the workers were anemic. These cases of acute lead poisoning were related to a change in progess involving the conversion of two small rotary furnaces previously used to process antimony to lead processing. Inadequate ventilation was felt to be responsible for the elevated air lead levels in the plant.

The median blood lead level of 38 workers at the secondary lead smelter at Eagan, Minnesota, was 72.5 μg/dl. Seventy-six percent had levels $\geq$ 60 μg/dl. Weakness of wrist and/or ankle extensor muscles was seen in 10.5 percent of the workers; 26 percent has tremor of the outstretched hands.

At the Atlanta, Georgia secondary lead smelter, 70 percent of the plants' 39 employees had blood lead levels $\geq$ 60 μg/dl. The percentage of workers with blood-lead levels $\geq$ 80 μg/dl varied between 43 percent in January, 1975 (when a lead exposure problem was first identified) to 26 percent in February 1976. Workers at this plant who were treated with intravenous chelation were permitted to remain on the job during therapy. This practice is condemned by the medical profession.

An important finding of the overall study was that blood lead and erythrocyte protoporphyrin (EP) were well correlated with symptoms of lead toxicity. Persons reporting symptoms during the year previous to testing had significantly higher blood lead levels than those without

TABLE 7.14 BLOOD LEAD LEVELS IN LEAD PLANT WORKERS[a]

Blood Lead Level, µg/dl	Number of Workers in Each Group, Percent at the Level Indicated					
	Memphis, Tenn.	Joplin, Mo.	Atlanta, Ga.	Salt Lake City, Utah	Eagan, Minn.	Total
< 40	14(18%)	1(2%)	6(15%)	1(3%)	3(8%)	25(11%)
40-59	12(15%)	7(17%)	6(15%)	5(17%)	6(16%)	36(16%)
60-79	26(33%)	21(50%)	17(44%)	2(7%)	17(45%)	83(37%)
≥ 80	26(33%)	13(31%)	10(26%)	21(72%)	12(32%)	82(36%)
Total Tested	78	42	39	29	38	226

[a] Source: Center for Disease Control (1977).

TABLE 7.15 PERCENTAGE OF WORKERS WITH SYMPTOMS OF LEAD POISONING BY BLOOD LEAD AND ERYTHROCYTE PROTOPORPHYRIN LEVEL AT THREE LEAD PLANTS[a,b]

Plant Location	Ratio, Percent, of Symptomatic Workers by Blood Lead Level, µg/dl			
	<40	40-59	60-79	>80
Tennessee	1/13 (8)	2/12 (17)	7/26 (27)	18/26 (69)
Missouri	[0/1 (8)][c]	4/7 (57)	7/21 (33)	7/13 (54)
Minnesota	[0/2 (0)]	2/6 (33)	9/14 (64)	5/12 (42)
	Erythrocyte Protoporphyrin Level, µg/dl			
	<100	100-199	200-299	>300
Tennessee	2/26 (8)	2/13 (15)	11/16 (69)	13/21 (62)
Missouri	[1/1 (100)]	4/10 (40)	10/15 (62)	9/15 (60)
Minnesota	[0/2 (0)]	1/5 (20)	[0/4 (0)]	15/23 (65)

[a] Source: Adapted from Center for Disease Control (1977).
[b] Two or more of symptoms of lead poisoning during past year.
[c] [ ] = 5 members or less.

symptoms. Erythrocyte protoporphyrin (EP) levels were also well correlated (r = 0.76) with zinc protoprophyrin levels. Furthermore, the higher the blood lead or EP level, the greater was the proportion of symptomatic workers. This correlation was best in circumstances where the duration of exposure was relatively short as in the Memphis facility (average duration of employment was 5 months) than in plants where the average duration of exposure was longer. In Eagan, Minnesota, and Joplin, Missouri, the average duration of exposure was 4 and 20 years respectively. These data are shown in Table 7.15.

Forty-nine percent of workers with blood-lead levels from 60-79 μg/dl reported symptoms consistent with lead poisoning during the year preceding examination. Twenty-three percent of workers with blood lead levels from 60-79 μg/dl were experiencing symptoms at the time of the examination. Since many of the symptoms reported were nonspecific, (i.e., headaches, joint pain, constipation) a similar questionnaire was administered to a control group of workers residing in the same community but without occupational lead exposure. The prevalence of reported symptoms consistent with lead toxicity was much lower among unexposed workers than in lead workers.

Anemia, (defined by hemoglobin levels < 14 g/dl) was noted in 14 percent of workers at the Memphis plant and 31 percent at the Salt Lake City facility. The clinical significance of anemia depends upon the degree of hemoglobin depression, however, classical symptoms of anemia (i.e., tiredness, fatigue, etc.) are not generally seen until hemoglobin decreases below 8 g/100 ml. Impairment in work performance, however, has been documented at hemoglobin levels of 11-13 g/dl according to the authors (Center for Disease Control, 1977). A dose-response effect between hemoglobin level and work performance was noted throughout the range of hemoglobin levels from 3-17 g/dl and no threshold effect was observed. Resistance to infection is also depressed in anemic persons although no precise information regarding the magnitude or threshold level for this effect is available.

The manifestations of lead toxicity varied with duration of exposure. After brief exposure (2-3 months) gastrointestinal symptoms predominated whereas constitutional symptoms and joint pains were relatively more common after prolonged exposure (over 1 year). Also, conditions of acute exposure led to relatively more gastrointestinal symptoms than chronically exposed workers. Highest rates of gastrointestinal symptoms were noted in lead poisoning cases in Salt Lake City (3-4 months duration) while workers chronically exposed in the Eagan, Minnesota, plant exhibited more nonspecific, constitutional symptoms such as tiredness, weakness, irritability, and joint pain (Tables 7.16 and 7.17).

A summary of dose-response data for various symptoms is as follows:

1. Anemia - With one exception, anemia was observed only after two or more months of exposure.
2. Neuropathy - Several workers showed wrist extensor or ankle weakness after 2-7 months of exposure. The lowest blood lead level associated with neuropathy was 78 μg/dl; most neuropathy cases had blood lead levels above 80 μg/dl.

TABLE 7.16 FREQUENCY OF SYMPTOMS AMONG 22 CASES OF LEAD POISONING, UTAH, 1976[a]

Symptom	Number	Percent
Abdominal cramps	19	86
Nausea	18	82
Diarrhea	12	55
Headache	12	55
Dizziness	12	55
Anorexia	12	55
Vomiting	9	41
Paresthesias	9	41
Constipation	8	36
Fatiguability	8	36
Myalgias	7	32
Weight loss	6	27
Irritability	5	23
Chest pain	5	23
Muscle weakness	4	18
Tremor	4	18
Joint pains	4	18

[a] Source: Adapted from Center for Disease Control (1977).

TABLE 7.17 FREQUENCY OF SYMPTOMS IN LEAD SMELTING PLANT EMPLOYEES, MINNESOTA, 1976[a]

Symptom	Number	Percent
Tiredness	17	52
Loose bowel movements	11	33
Irritability	9	27
Joint pains	9	27
Muscle weakness	9	27
Loss of appetite	7	21
Headache	6	18
Leg cramps	6	18
Constipation	6	18
Nausea[b]	5	16
Trouble sleeping	4	12
Vomiting	4	13
Abdominal pain	3	9
Weight loss[c]	2	6
Shakes	2	6

[a] Source: Adapted from Center for Disease Control (1977).

[b] Excluding 1 employee with symptom due to ulcers.

[c] Excluding 2 employees with symptom due to diet. Of the 2 with weight loss not related to diet, 1 lost 20 pounds in 15 months and 1 lost 22 pounds in 7 months.

3. Nephropathy - No definite relationships were noted between duration of exposure and development of overt lead nephropathy. Some abnormalities of renal function were correlated with duration of lead exposure. At the Missouri plant, 32 percent of workers tested had elevated blood urea nitrogen (BUN). These workers had worked at the plant for 45 - 31 years. Eight percent of the Tennessee workers had elevated BUN levels and 8 percent of the Minnesota workers had elevated serum creatinine levels. With the exception of three individuals, (all of whom had been exposed to lead less than two months) no worker with less than 3 1/2 years of exposure had abnormal renal functions tests.

A survey of 228 children living in the area surrounding the Memphis, Tennessee smelter showed no excessive lead absorption, however young children of smelter workers were found to have elevated blood levels, with eight of these children being hospitalized and treated for lead poisoning. Examination of 31 children (ages 1-9) at the Eagan, Minnesota plant revealed blood lead levels $\geq$ 30 μg/dl in 16 percent of the children, indicating increased lead absorption.

Based on findings presented in the previous discussion, it is obvious that the currently accepted guideline defining the upper limit of a safe blood lead level (80 μg/dl) does not provide adequate protection against lead toxicity. If lead toxicity is to be prevented, the biologic monitoring system must effectively remove the worker from exposure prior to the onset of illness. Clearly, the present 80 μg/dl level does not provide adequate protection since anemia and other overt symptoms of lead intoxication were observed at blood-lead levels below 80 μg/dl.

In the five facilities investigated, no workers showed evidence of lead related anemia, wrist weakness or lead related renal disease associated with a blood lead level < 60 μg/dl. In fact, very few workers with blood lead levels < 60 μg/100 ml had any symptoms consistent with lead intoxication. It was concluded that the data in the report clearly support the setting of a biologic threshold limit value of 60 μg/dl for occupationally exposed populations, and furthermore, that this value should be revised downward in the event that deleterious health effects at blood lead levels < 60 μg/dl can be demonstrated in the future (Center for Disease Control, 1977).

7.5.3.1.2 Effects of chronic lead exposure on mortality--Yet another possible means of investigating clinical effects of lead exposure is the systematic examination of mortality patterns of lead workers. Customarily such studies are retrospective in nature and involve a comparison of the mortality rates for various causes among lead workers as opposed to "control groups" having no unusual sources of lead exposure (i.e., general populations). Findings of elevated rates or proportions of various causes of deaths among lead workers suggests that lead may play a role in the etiology of that particular disease. Although in general, occupational mortality studies of the lead industry have produced essentially negative results, several such investigations are summarized below.

Dingwall-Fordyce and Lane (1963) carried out a retrospective mortality study of British lead workers (with at least 25 years of service each) who retired between the years 1926 and 1960. Workers were classified according to three grades of exposure: heavy, medium or none based on average urinary lead concentration records. The heavy exposure group (average urinary lead 100-250 μg/liter over the past 20 years of employment) had higher than normal death rates from cerebrovascular diseases. Included in this category are cerebral hemorrhage, thrombosis and arteriosclerosis. Interestingly, the trend of excess deaths in the heavy exposure group was most pronounced among men who retired prior to 1951. Such a finding is probably related to technological changes which improved exposure conditions over the years. Deaths from malignant neoplasms were not above the rate expected (based on the general population rates) in any of the exposure categories.

Malcolm (1971) also published the results of a mortality study which examined men exposed to lead at "moderate" levels. The average blood lead level of the group was 65 μg/dl. He found no evidence of significant excess mortality from heart disease, pulmonary disease, cerebrovascular accidents, cancer, renal disease or other miscellaneous causes among the group.

Cooper and Gaffey (1975) undertook a study of men who worked in lead production facilities and battery plants to determine whether or not their mortality differed significantly from that of men not occupationally exposed to lead. This investigation took the form of a prospective study of mortality in six lead-production facilities and ten battery plants. The former included one primary smelter, two refineries, and three recycling plants. A group of 7032 men who had been employed in these plants between 1946 and 1970 was followed to determine whether they were still alive at the end of 1970. Their observed mortality by cause was compared with that of a comparable population of all United States males.

Lead absorption in many of the men was greatly in excess of currently accepted standards based upon urinary and blood-lead concentrations available for a portion of the group. Seventy-eight percent of 47 smelter workers had blood lead levels $\geq$ 80 μg/dl or more over the period from 1946-1961. After 1965 data based on 489 workers showed 35 percent had blood lead $\geq$ 80 μg/dl. Levels from the battery workers were somewhat lower than those of the smelter workers. Over all levels of exposure, duration and job classifications, 81.5 - 95.7 percent of the workers had blood lead levels $\geq$ 40 μg/dl.

There were 1356 deaths reported; death certificates were obtained for 1267. The standardized mortality ratio (SMR) for all causes was 107 for smelter workers and 99 for battery plant workers indicating a fairly close approximation of the U.S. male population whose SMR = 100 by definition. Deaths from neoplasms were in slight excess in smelters, but not significantly increased in battery plants. There were no excess deaths from kidney tumors. The SMR for cardiovascular-renal disease was 96 for smelter workers and 101 for battery plant workers, i.e., roughly the same as for the general population but not as good as might be expected in an employed population which commonly have lower SMRs for these causes than the general population. There was definitely no excess in deaths from either stroke or hypertensive heart disease. However, deaths classified as "other hypertensive disease" and "unspecified nephritis or renal sclerosis" were higher than expected. The actual numbers of deaths in these last-named categories combined (41 where 19.5 were expected) represented about three percent of all certified deaths. In these observations, the possible interactive effects between lead and other renal toxic agents, such as cadmium cannot be ruled out, however. The life expectancy of lead workers was calculated to be approximately the same as that of all U.S. males.

7.5.3.1.3 Subclinical effects: hematologic, neurologic, behavioral, and nephrotoxic--As noted above, lead may produce changes in biochemical and physiological parameters which occur at blood-lead levels well below those usually associated with overt clinical effects. The point at which subclinical changes became sufficiently serious to represent a threat to health are neither clearly defined nor agreed upon.

Selander and Cramer (1970) and Haeger-Aronsen (1971) have reported that the threshold level associated with the appearance of excess ALA in the urine occurs at blood lead levels in the 40-60 μg/dl range. An exponential increase in urinary ALA (reflecting inhibition of ALAD by lead) is seen as blood lead increases above 40 μg/dl. The physiological significance of this disturbance of heme synthesis is that if sufficient inhibition occurs, overt hematological changes such as anemia, and decreased life span of erythrocytes may result.

Similarly, Tola, et al., (1973) reported a slight decrement in hemoglobin and hematocrit at blood-lead levels as low as 40 μg/dl. Their conclusion was based on a study of sequential changes in hemoglobin which occurred among workers newly employed in an industrial lead environment.

Studies by Seppalainen, et al., (1975) revealed subclinical neuropathy (slowing of the maximal conduction velocities of the median and ulnar nerves) in storage battery workers whose blood lead levels never exceeded 70 μg/dl. These changes may be precursors of more serious motor impairment in view of the fact that these effects may represent a milder version of the weakness of the extensors of the hands and feet which is frequently observed in clinical lead intoxication. Subtle effects of the nervous system take on added significance due to the possible irreversibility of these changes and the limited regenerative capacity of the nervous system.

The data of Seppalainen, et al., (1975) agree reasonably well with those of Repko, et al., (1975), who studied behavioral measures of task performance among workers exposed to lead in storage-battery manufacturing companies. While the intellectual functions tested were unaffected by increases in body burden of lead, motor functions (i.e., hands), sensory functions (hearing), neuromuscular or psychomotor (tremor, eye-hand coordination, muscular strength, and endurance) and psychological functions (as measured by tests of hostility, aggression, and general dysphoria) were all affected by the body burden of lead. The strongest relationships between exposure and effects were found with tests of neuromuscular and psychomotor performance. Major changes on the preferred side of the body were observed at blood levels between 70 and 79 μg/100g (U.S. Department of Labor, 1975).

Vitale, et al., (1975) performed various laboratory/diagnostic tests of renal function on a group of lead workers. The tests revealed evidence of decreased renal function in 4 of 8 persons with blood lead levels below 80 μg/dl. These lead workers had been engaged in refining, welding and cutting operations for variable lengths of time. The functional impairment associated with this level of renal damage, if any, was not reported. Similarly, Cramer, et al., (1974) observed renal damage including decreased ability of proximal tubular cells to form inclusion bodies, decreased ability to excrete lead and moderate renal fibrosis in a group of workers exposed to lead for 4 years or longer.

7.5.3.1.4 Policy implications of air to blood lead relationships for setting occupational standards--A variety of recent studies have generated data on the dose-response relationship between air lead and blood lead. Since these studies are more relevant from the standpoint of implications for standard setting (rather than documenting specific forms of toxicity which occur at a given lead level) they are reported separately in the section below. In reading this section, it is well to keep in mind that most industrial firms use blood lead as an indirect measure "dose" of air lead, rather than direct monitoring of air lead, which may vary substantially throughout the plant. Second, as reported in the previous sections, blood lead levels are well correlated with a variety of health effects, both clinical and subclinical. Hence periodic monitoring of blood lead levels may be quite useful in preventing overexposure or failing that, in detecting lead effects at an early and often reversible stage.

Preliminary findings of a study by National Institute for Occupational Safety and Health (NIOSH) at the General Motors Corp. Delco-Remy battery plant in Muncie, Indiana, provide new information on the relationship between air and blood-lead levels (Wall Street Journal, 1977b). NIOSH analyzed the data on lead levels in the blood of about 500 plant workers as well as the lead level in the air. Investigators found that the plant kept lead levels in the blood of over 90 percent of its workers at 60 μg/dl or lower by maintaining air exposures at a maximum of about 100 μg/m^3 in most work areas.

The studies of Williams, et al., (1969) and Williams (1972) cited in U.S. Department of Labor (1975) may be used to estimate the potential contribution of lead in air to blood lead levels. The air exposure data used by Williams, et al., (1969) was based on measurements obtained from workers in various departments of a storage battery factory who wore personal samplers for a full work shift for 2 weeks. Considerable variation was found both among departments and among individual personal samples. The study presents a correlation between the mean blood lead level, together with the upper and lower ranges, with air lead levels. In order to provide a maximum blood lead value of 60 μg/dl, the mean blood lead level in a population of workers must be maintained at about 40 μg/dl since a mean of about 40 μg/dl will result in a range in workers of approximately 20 μg/dl at the lower limits to 60 μg/dl at the upper limits. The Williams data suggest that air lead levels of 200 μg/m^3 correspond to a range of 48-92 μg/dl of lead in blood with a mean level of 70 μg/dl (84 μg/dl ≈ 80 μg/100g). Air lead levels of 150 μg/m^3 correspond to a range of 38-82 μg/dl with a mean blood lead level of 60 μg/dl. It is apparent, therefore, because of these ranges, that airborne lead levels of 200 or even 150 $\mu g/m^3$ would result in unacceptable blood lead levels, i.e., greater than 60 μg/dl for a significant number of employees (U.S. Department of Labor, 1975).

Based on data from 103 employees, air lead levels of 200 $\mu g/m^3$ produced a mean blood level of 70 μg/dl with some workers having over 80 μg lead/dl in blood (Williams, 1975). Interestingly, the data on air lead and corresponding blood lead levels reported by Short Associates (1976) tends to confirm the relationship described above.

The correlation of somewhat lower blood lead levels, (i.e., < 60 μg/dl) with air lead levels are not definitive. Thus, the regression equation relied upon in Williams' study is based upon data which do not contain a critically important value--the blood lead level corresponding to an air lead level of 50 $\mu g/m^3$. If it is assumed that the Williams regression equation is correct, an air lead level of 50 $\mu g/m^3$ would correspond to a mean blood lead level of 40 μg/100g, with an upper limit of approximately 60 μg/100g. However, as noted above, critical data are missing, leaving doubt as to the correctness of this projection. The regression equation is unduly weighted by data at either extreme, and extrapolation of this equation to predict the air lead-blood-lead relationship beyond the limits of measured data is somewhat speculative. In any event, the study by Williams, even with its limitations, provides the best available data with respect to the air lead--blood lead relationship in industrial exposure situations. Clearly, the Williams study shows that to achieve a mean blood lead level of 40 μg/100g would require an 8-hour air lead concentration of less than 150 $\mu g/m^3$ based on a time-weighted average (U.S. Department of Labor, 1975). Based on Williams' (1969) calculations, it appears that an increase of 1 $\mu g/m^3$ in the weekly time-weighted average concentration of lead in air would correspond to an increase of approximately 1 μg/dl in blood lead.

Prpic-Majec, et al., 1973 and Fugas, et al., 1973, report data from a study of 52 workers in unspecified lead trades. The time-weighted average concentration of respirable lead particles was 35 $\mu g/m^3$; the mean blood lead level of the group was 44.3 μg/dl. A control group living in an area having air lead at 0.2 $\mu g/m^3$ and a mean blood lead level of 22.4 μg/dl was used to establish baseline levels of blood lead due to non-air sources of 22.1 μg/dl (0.3 μg/dl in the control group was due to air). Since the air lead level in the occupational environment was 35 $\mu g/m^3$, it was concluded that 1 μg of lead mer m^3 in air contributes 0.6 μg/100 ml of blood (22.1 $\mu g/m^3 \div 35 \mu g/m^3$).

As earlier indicated, the studies of Prpic - Majec, et al., (1973) Fugas, et al., (1973) and Williams (1975) suggest that the incremental increase in blood lead per unit increase in lead in air is somewhat less at air lead levels characteristic of industry than those in the usual outdoor ambient range.

7.6 ORGANIC LEAD

In general, the alkyl derivatives of lead are highly toxic compounds, and are readily absorbed through the skin. Tetraethyl and tetramethyl (TEL and TML) are clear, colorless liquids, volatile, nonpolar, nonionic, and soluble in many organic solvents such as hydrocarbons, chloroform, ether, and absolute ethanol. They have very low water solubilities, and are relatively unreactive in air, water or alkali (Shapiro and Frey, 1968). The high solubility of alkyl lead compounds in body fluids is the basis for toxicity through percutaneous absorption whereas the volatility of these compounds (particularly when added to gasoline) underlies the hazard potentail among workers engaged in the production of these compounds. Those chronically exposed to evaporating gasoline may also be at risk, although few studies have been conducted. Because alkyl lead compounds are light-sensitive and undergo photochemical decomposition when they reach the atmosphere; their presence in the atmosphere is transient (National Academy os Sciences, 1972).

In cities, alkyl lead levels were reported "not to reach 10 percent of inorganic levels" (National Academy of Science, 1972). In fact, much of the lead burned in gasoline (see Section 6.6.1) is not exhausted in forms which can remain suspended in the atmosphere. To date, no information exists on the effects of chronic, low-level exposure to airborne alkyl lead.

From a toxicological viewpoint, the most significant organic lead compounds are the lead alkyls, tetraetheyl and tetramethyl. Additionally, a number of lead ompounds of organic acids, such as lead naphthenate, lead octoate, etc., used in the paints and plastics industry are significant although these are, by industry convention, classified as inorganic compounds. Their behavior in biological systems also appears more similar to inorganic lead than to that of the lead alkyls.

Lead soaps of organic acids are commonly used as driers by the paint, varnish, printing ink and linoleum industries. Thus, exposure to these compounds is limited mostly to industrial workers. Soaps of mixed acids of linseed oil and rosin acids are known as linoleate driers and resinate driers, respectively. Lead stearates, tallates, naphthenates, and octoates (chiefly 2-ethylhexanoate) are also produced and marketed in the United States as paint driers. The hazards encountered in the handling and use of driers and metallic soaps are associated with the toxicity of the metal present, the solvents contained, and their activity as oxidation catalysts. Although little data are available regarding the specific toxicities of these compounds, in general, the LD_{50} values appear to be several orders of magnitude higher than those of TEL and TML.

In general, the alkyl (tri-and tetraalkyl) derivatives of lead are highly toxic compounds which are absorbed by inhalation, ingestion or percutaneous absorption. These compounds are probably distributed in a nonionic form and, being lipid-soluble, are concentrated in the brain, body fat and liver. Because of this selective distribution, manifestations of lead poisoning are dominated by involvement of the central nervous system (CNS) and differ from those of inorganic lead poisoning (Gerarde, 1964; Schepers, 1964). While many of the symptoms of acute poisoning by inorganic and organic lead are similar, they can be distinguished diagnostically. Organolead poisoning results in elevated levels of lead in the urine but less consistently in the blood. Likewise, red blood cell stippling, anemia, and alterations in urinary porphyrins are not consistently seen with organic lead exposure, particularly in acute episodes.

Organic lead compounds, particularly the two alkyl leads used in antiknock additives, constitute primarily an occupational exposure problem, to which the general population is not exposed. Because of the known occupational hazard, extensive studies have been conducted on uptake and absorption, elimination, tissue distribution, and toxic effects. The results of these studies are summarized briefly in the following pages. A more detailed discussion of the health effects of organic lead compounds is contained in the recent review of the environmental effects of lead (Bell, et al., 1978).

7.6.1 Uptake and Absorption

Kehoe (1925, 1927) showed that tetraethyllead vapor is readily absorbed through the pulmonary epithelium. Using radium-D as a marker, Mortensen (1942) studied the uptake of tetraethyllead in the lungs of rats. The amount absorbed was proportional to the concentration of vapor in the inhaled air, although the proportion decreased at high concentrations (Mortensen, 1942). Because lead alkyls are broken down by light and heat, their presence in the air is transient and it is believed that they contribute little to the inhalation burden of general populations.

Ingestion is a much less probable route for uptake and absorption of organic lead compounds than inhalation and skin absorption. In general, lead soaps are hydrolyzed and possibly converted to metal chlorides and free acids when ingested. Hazards from inhalation and skin contact may also arise when solvents are combined with the lead soaps, particularly in the liquid driers. In such situations, the solvents probably represent a greater toxicological hazard than the lead in the paint driers. One estimate places the total number of persons directly exposed to lead in the manufacture of paint driers in the United States at less than 250; thus, it is a relatively insignificant source of lead exposure compared to the primary and secondary lead and storage battery industries which employ about 25,000 persons who are potentially exposed to much higher levels (Short Associates, 1976). Further, the usage of many types of paint driers, especially the linoleates, resinates, stearates, and tallates has markedly declined in the past decade, further reducing any possible hazard.

Pettinati, et al., (1959) and Rosetti, et al., (1961) point out that percutaneous absorption is of importance only in the case of lead alkyls and lead salts of naphthenic and fatty acids. Eldridge (1924) reported that tetraethyllead was absorbed rapidly through the skin in both dogs and guinea pigs.

Laug and Kunze (1948) found the amount of lead in the kidney from application of tetraethyllead was significantly elevated relative to the controls who received various forms of inorganic lead, demonstrating the greater percuteneous absorption of tetraethyllead. (Table 7.18).

Table 7.18 CUTANEOUS ABSORPTION OF LEAD COMPOUNDS[a]

Compound	Quantity Applied, mg/g	Lead Concentration in Kidneys, Wet Wt.	Control
Arsenate	102	0.85	0.55
Oleate	148	1.30	0.59
Acetate	77	1.80	0.82
Tetraethyl	106	64.20	0.82

[a] Source: Laug and Kunze (1948). Reprinted, with permission, from J. Ind. Hygiene Toxicol.(c) Williams and Wilkins Co.(1948).

Tetraalkyl lead compounds are volatile, highly lipid-soluble, and would be expected to pass through biological membranes including the placenta. McClain and Becker (1972) reported that trimethyllead does cross the placental barrier but to only a limited extent at low maternal doses. At higher doses, however, transport was much greater, probably because binding sites in maternal erythrocytes were saturated at the higher dose.

7.6.2 Tissue Distribution

The distribution of the organic lead salts differs markedly from the inorganic salts of lead. Bolanowska (1968) investigated the distribution and excretion of triethyllead in rats following intravenous administration of tetraethyllead. Twenty-four hours after administration of tetraethyllead, 50 percent of the total lead in the internal organs was in the form of triethyllead; the highest levels were found in the liver, blood, kidney, and brain. Cremer (1959) found that tetraethyllead was rapidly converted to triethyllead in the liver. Triethyllead then remained stable *in vivo* for several days before it was excreted in the feces and urine at a rate equivalent to not more than 1 percent of the daily dose. Inorganic lead constituded the remainder of the *in vivo* lead beginning 24 hours after injection.

The triethyllead ion is identified as responsible for the toxic effects of tetraethyllead. There is evidence that tetramethyl- and tetrapropyllead compounds are metabolized to the trialkyl form much more slowly than tetraethyllead is converted to triethyllead. This may account for their being comparatively less toxic, although once formed, trimethyllead compounds are as toxic as the triethyllead compounds (Cremer and Callaway, 1961). Unlike those of inorganic lead, the organic lead compounds have no special affinity for bone but a high affinity for lipid tissues, especially those constituting the nervous system. Therefore, the central nervous system is the site of greatest concentration.

Schepers (1964) observed that the concentration in the brain is much lower than in the liver and lungs, even though the effects of organic lead compounds are mainly in the central nervous system in experimental animals. After inhalation of toxic amounts of tetraethyllead the concentration of lead in the skeleton and in blood is negligible, whereas after inhalation of toxic amounts of tetramethyllead, the concentration of lead in blood may exceed that in any other tissue including the skeleton. Following inhalation by rats, tetraethyllead can be recovered unchanged from the liver (Stevens, et al., 1956), although it is rapidly metabolized in the liver to inorganic lead so that chronic exposure may result in distribution and a toxic syndrome characteristic of inorganic lead (Cremer, 1959).

7.6.3 Elimination

There are few data on the excretion of alkyl lead compounds such as tetraethyllead and tetramethyllead. Sanders (1964) states that in both inorganic and organic lead intoxication the urine will probably be found to have an abnormally high concentration of lead. In tetraethyllead intoxication, it is likely to be considerably higher than it is an inorganic lead poisoning since lead absorbed percutaneously is more rapidly excreted.

In considering the kinetics of organic lead excretion, it should be noted that TEL and TML have a far shorter residence time in the blood and other tissues than does inorganic lead. Kehoe and Thamann (1931) found that in rabbits, following skin absorption, the absorbed tetraethyllead decomposed in the tissues after a period of 3 to 14 days. Both its tissue distribution and excretion followed quantitatively that of water-soluble lead compounds. In a study of the excretion of triethyllead in rats, Bolanowska (1968) found that 24 hours after the administration of TEL, 50 percent of the total lead in the soft organs was in the form of triethyllead. Triethyllead concentration *in vivo* then remained steady for several days, and was excreted in the feces and urine at a rate equivalent to not more than 1 percent of the daily dose of TEL. Inorganic lead constituted the remainder of the *in vivo* lead 24 hours after TEL injection, and no diethyllead was found, even shortly after administration of TEL. Diethyllead was fairly stable *in vivo*, but less so than triethyllead. Bolanowska notes the presence of triethyllead in humans who died up to 20 days after acute poisoning with TEL.

7.6.4 Toxic Effects

7.6.4.1 Acute Toxicity of Various Organolead Compounds--

The absorption of lipid-soluble alkyl lead compounds, either through the skin or the lungs may result in central nervous system manifestations including insomnia, asthenia, tremors, headache, neuromuscular pain, hallucinations, mania, delusions, and convulsions. The results of the administration of single, lethal doses of TEL or TML progress from hyperirritability and tremors to convulsions and death. Chronic exposure to alkyl lead also leads to neurotoxicity with similar symptomatology as that following acute exposure.

The toxicities of TEL and TML are shown in Table 7.19. As shown, the TLV's (Threshold Limit Values) for lead as tetramethyl lead is 100 μg/m^3, and 150 μg/m^3 for tetraethyl lead. These are, however, for skin exposure, not for inhalation.

7.6.4.2 Neuropathology--

The pathological lesions noted in lead induced CNS anomalies include cerebral edema, encephalopathy, increased cerebrospinal fluid pressure, proliferation and swelling of endothelial cells, dilation of capillaries and arterioles, glial cell proliferation, focal necrosis and neuronal degeneration (Goyer and Rhyne, 1973).

Many of these symptoms are also common to inorganic lead poisoning as well.

One investigator has attempted to distinguish between CNS toxicity caused by organic and inorganic lead compounds. This is the monograph prepared by Pentschew (1958) based principally on earlier work of Tolgskaya and Reznikov (1955). These authors discussed the physiologic differences between encephalopathy caused by organic lead compounds versus that attributable to inorganic lead.

Triethyllead inhibits catecholamine effect on smooth muscle contraction. Consequently, there has been some speculation that the toxicity of triethyllead and particularly its action on the central nervous system can be explained by a combination of effects which might result from an upset of cholinergic and adrenergic contral pathways due to the formation of endogenous psychotogenic complexes. This speculation is based on unconfirmed claims that adrenochrome, which is an intermediate in the conversion of catecholamines to trihydroxyindole products, is an hallucinogenic agent. Galzigna, et al., (1973) found that triethyllead dramatically increases the duration of the succinylcholine-induced myoneural block of cholinergic transmission. This is explained by the competition between triethyllead and succinylcholine for serum cholinesterase. Triethyllead is able to antagonize the affect of norepinephrine _in vivo_ but alone is without any effect on normal contractile activity.

7.6.4.3 Metabolic Effects--

Few studies have focused specifically on the metabolic effects of alkyllead compounds.

Trimethyl-and triethyllead decrease oxygen consumption when added to rat brain and cortex slices _in vitro_. Tetramethyl- and tetraethyllead did not have this effect except at much higher concentrations (amount unknown). A decrease in _in vitro_ oxygen consumption was also found in brain slices taken from rats previously exposed to triethyl- and tetraethyllead (Gerarde, 1964). Glucose metabolism in brain slices is also inhibited by the diethyl-, triethyl-, tripropyl- and trimethyllead compounds.

TABLE 7.19 REPRESENTATIVE TOXICITIES AND OCCUPATIONAL STANDARDS FOR TETRAETHYL LEAD AND TETRAMETHYL LEAD[a]

Compound and Chemical Formula	Standards, μg/m^3		Toxicities, mg/kg[d]		
	U.S. Occupational[b]	TLV[c]			
Tetraethyl lead $Pb(C_2H_5)_4$	75 (skn)	100 (skn)	oral rat	LD_{Lo}	17
			ipr rat	LD_{Lo}	10
			ivn rat	LD_{Lo}	31
			par rat	LD_{Lo}	15
			scu mouse	LD_{Lo}	86
			scu mouse	TD_{Lo}	86
			skn dog	LD_{Lo}	500
			ivn rabbit	LD_{Lo}	23
			skn guinea pig	LD_{Lo}	990
			inh rat	LC_{50}	6ppm
Tetramethyl lead $Pb(CH_3)_4$	70 (skn)	150 (skn)	oral rat	LD_{50}	190
			ipr rat	LD_{Lo}	73
			par rat	LD_{50}	105
			ivn rabbit	LD_{Lo}	90

Notes:

[a] Source: Registry of Toxic Effects of Chemical Substances (NIOSH, 1977).
Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment (ACGIH, 1977)
U.S. Department of Labor (1978).

[b] U.S. Occupational Standard (OSHA) based on time-weighted average (TWA) for continuous exposure (8-hr day and 40-hr week).

[c] Threshold Limit Value - based on time-weighted average for continuous exposure 8-hr day and 40-hr week (American Conference of Governmental Industrial Hygienists (1977).

[d] Toxicity data descriptors: See Table 7.7

TEL has effects on neurotransmitter metabolism in the brain of experimental animals (Cremer and Callaway, 1961). The mechanism of this action was studied by assessing the effects of triethyllead *in vitro* on the cholinesterase activity of rat diaphragm and *in vivo* on serum cholinesterase in the dog. The highest inhibition of acetylcholinesterase activity of the rat diaphragm obtained with two concentrations (0.4 and 0.8 milliliter) of the substrate acetylthiocholine was about 25 percent of normal activity. The inhibition was noncompetitive. As with other aspecific cholinesterase inhibitors, triethyllead was able to induce a maximal inhibition of 25 to 30 percent on the true cholinesterase activity of the rat diaphragm preparation. Maximum inhibition of serum cholinesterase was observed 15 minutes after the administration of 6 milligrams per kilogram triethyllead to dogs and full recovery occurred 45 minutes after administration (Galzigna, et al., 1973).

7.6.4.4 Enzyme Effects--

In view of the high sensitivity of ALAD to lead, the levels of enzyme activity in the blood of men occupationally exposed to lead alkyls, particularly tetraethyllead, were measured (Millar, et al., 1972). The enzyme activity in an exposed group of men was significantly less (P less than 0.001) than in a control group, the respective mean values being 220 and 677 units of enzyme activity.

Tetraethyllead is metabolized in the body via triethyllead and diethyllead ions. Diethyllead ion was found to inhibit ALA dehydratase activity at concentrations greater than 5×10^{-5} M, although the degree of inhibition was less than that obtained with Pb^{++}. These results suggest that exposure to tetraethyllead can cause a decrease in erythrocyte ALA dehydratase activity (Millar, et al., 1972). The effect of known inhibitors on ALA dehydratase activity, such as alcohol consumption and cigarette smoking were not taken into account in the group of workers investigated.

7.6.4.5 Reproductive Effects--

McClain and Becker (1972) found that tetraethyl lead, tetramethyl lead, and trimethylead chloride were essentially nonteratogenic in Sprague-Dawley rats. Embryo or fetal toxicity accompanied the administration of the organolead compounds and was characterized by growth retardation and delayed ossification of bone. Marked fetal effects were observed only in maternal animals that exhibited severe toxicity and were, therefore, severely debilitated.

7.6.5 Epidemiologic Studies

Gerarde (1964) states that because of the large dilution factor (3 to 4 milliliters per gallon of gasoline) of TEL, the normal use of "leaded" gasoline does not present a lead intoxication hazard. However, human exposure to TEL during the cleaning of leaded-gasoline tanks has led to fatalities (Shapiro and Frey, 1968). Robinson (1974) has presented evidence of absorption of alkyl lead antiknock compounds among occupationally exposed workers. Delta-aminolevulinic acid (ALA) and lead were measured in the urine of 123 men of various ages and lengths of service, all working as operators or maintenance men in an area of production of alkyl lead anti-knock compounds (tetraethyllead and tetramethyllead). The levels of ALA and lead in urine were found to have a relatively low positive correlation of 0.52 which suggests that a given elevation of urinary lead excretion probably is associated with a lower level of urinary ALA excretion when the exposure is to organic lead. It is not clear whether this difference might be related primarily to differences in circumstances of exposure or to differences in metabolism (biotransformation) due to exposure to a different form of lead. For example, there have been no experimental studies reported to indicate what effect, if any, tetraethyllead or tetramethyllead might have on porphyrin metabolism and, hence, on urinary ALA excretion. In summary, the investigation by Robinson (1974) shows that absorption of organic lead as indicated by increased urinary levels, is associated with an increase in urinary ALA levels. However, the mechanism through which this effect is produced was not identified.

Proper protection of workers has virtually eliminated the problem of TEL or TML poisoning over the last 30 years. Robinson (1976) made a detailed comparison of extensive medical information (results of periodic medical examinations, of medical records of absence from work due to nonoccupational illness, and of cumulative medical diagnoses) for workers with 20 years or more tetraethyllead (TEL) service to a matched (for age, sex, race, and length of service) group of workers with no occupational lead (TEL or other) exposure. The comparison showed no significant health differences between the two populations. Robinson (1976) concluded that under the conditions that have existed at the Baton Rouge, Louisiana alkyllead plant, workers having long occupational exposure to levels of lead (chiefly TEL) sharply in excess of that of the general public, but within a range termed "safe" by current industrial medical standards, have not suffered detectable impairment of health.

7.7 REFERENCES

Albert, R. E., R. E. Shore, A. J. Sayers, C. Strehlow, T. J. Kneip, B. S. Pasternach, A. J. Friedhaff, F. Covan, and J. A. Amino. 1975. Follow-up of children overexposed to lead. Environ. Health Perspect. Exptl. Issue No. 7:33-39 May.

Alexander, F. W., H. T. Delves, and B. E. Clayton. 1973. The Uptake and Excretion of Children of Lead and Other Contaminants. In: Proceedings, International Symposium, Environmental Health Aspects of Lead. Amsterdam, October 2-6, 1972. Organized by Commission of European Communities and the U. S. EPA. Commission of the European Communities Center for Information and Documentation. Luxembourg. pp. 319-331.

Allcroft, R. 1950. Distribution of Lead in the Tissure of Bovines After Ingestion of Various Lead Compounds. J. Comp. Pathol. 60:190-208.

American Conference of Governmental Industrial Hygienists. 1977. Threshold Limit Values (TLV's) for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1977. Cincinnati, Ohio. 94 pp.

Angle, C. R., and M. S. McIntire. 1974. Lead in Air, Dustfall, Soil, House-dust, Milk and Water: Correlation with Blood Lead of Urban and Suburban Children. Proceedings of 8th Annual Conference of Trace Substances in Environmental Health, Columbia, Missouri, June 11.

Anon. 1975. Scientists Find Increased Lung Cancer in Areas around Lead, Zinc Smelters. Toxic Materials News, 2(16):123

Aub, J. C., L. T. Fairhall, A. Minot, and P. Reenikoff. 1925. Lead Poisoning. Medicine 4:1

Azar, A., R. D. Snee, and K. Habibi. 1973. Relationship of Community Levels of Air Lead and Indices of Lead Absorption. In: Proc., Int. Symp., Environ. Health Aspects of Lead, Amsterdam, The Netherlands, October 2-6, 1972, D. Barth, A. Berlin, R. Engel, P. Recht, and J. Smeets (Eds.), pp. 581-594.

Azar, A., R. D. Snee, and K. Habibi. 1975. An Epidemiologic Approach to Community Air Lead Exposure Using Personal Air Samplers. In: Lead; Environmental Quality and Safety Supplement, Volume 2, T. B. Griffin and J. H. Knelsen, (Eds.) Georg. Thienil, Publishers, Stuttgart.

Barltrop, D. 1969. Transfer of Lead to Human Foetus. In: Mineral Metabolism in Paediatrics, S. Garltrop, and W. L. Burland, (Eds.). Blackwell Scientific Publications, Oxford. 135 pp.

Barltrop, D., and A. Smith. 1971. Interaction of Lead with Erythrocytes. Experentia. 27(1):92-93.

Barry, P. S. I. 1973. A Comparison of Lead Concentrations in Human Bones and in Soft Tissues. In: Proc. of Int. Symp., Environ. Health Aspects of Lead, Amsterdam, October 2-6, 1972. D. Barth, A. Berlin, R. Engel, P. Recht, and J. Smeets, (Eds.). pp. 415-426.

Barry, P. S. I., and D. B. Mossman. 1970. Lead Concentrations in Human Tissues. Brit. J. Ind. Med. 27:339-351.

Bauchinger, M., E. Schmid, and D. Schmidt. 1972. Chromosomenanalyse bei Verkehrsoplizisten mit erhohter Bleilast. Mutat. Res., 16: 407-412.

Bauchinger, M., E. Schmid, H. J. Einbrodt, and J. Dresp. 1976. Chromosome Aberrations in Lymposytes after Occupational Exposure to Lead and Cadmium. Mutat. Res. 40:57-62.

Baumslag, N. 1975. Lead Poisoning. Lancet. 1(7916):1145.

Beattie, A. D., M. R. Moore, A. Goldberg, M. J. W. Finlayson, J. F. Graham, T. M. Machie, J. C. Main, P. A. McLaren, K. M. Murdock, and F. T. Stewart. 1975. Role of Chronic Low-Level Lead Exposure in the Aetiology of Mental Retardation. Lancet. 1:589-592, 1975.

Bell, M. A., R. A. Ewing, G. A. Lutz, V. L. Holoman, B. Paris, and H. H. Krause. 1978. Reviews of the Environmental Effects of Pollutants. VII. Lead. EPA 600/1-78-029. U. S. Environmental Protection Agency, Health Effects Research Laboratory, Cincinnati, Ohio. Various pagination.

Benson, P. F., and J. J. Chisolm, Jr. 1960. A Reliable Qualitative Urine Coproporphyrin Test for Lead Intoxication in Young Children. J. Pediat. 56:759-767.

Betts, P. R., Astley, R., and Raine, D. N. Lead Intoxication in Children in Birmingham. Brit. Med. J. 1:402-406, 1973.

Bingham, E. 1970. Trace Amounts of Lead in the Lung. In: Trace Substances in Environmental Health - III, Proc. 3rd Ann. Conf. on Trace Substances in Environ. Health, University of Missouri, Columbia, Mo., June 24-26, 1969. D. D. Hemphill, (Ed.) pp.83-90.

Blaxter, K. L. 1950. Lead as a Nutritional Hazard to Farm Livestock, II. The Absorption and Excretion of Lead by Sheep and Rabbits. J. Comp. Pathol. 60:140-159.

Blaxter, K. L., and A. T. Cowie. 1946. Excretion of Lead in the Bile. Nature 157(3992):588.

Bolanowska, W. 1968. Distribution and Excretion of Triethyllead in Rats. Brit. J. Ind. Med. 25:203-208.

Butt, E. M., R. E. Nusbaum, T. C. Gilmour, S. L. Dido, and S. Mariano. 1964. Trace Metal Levels in Human Serum and Blood. Arch. Environ. Health 8(1): 52-57.

Castellino, N., and S. Aloj. 1964. Kinetics of the Distribution and Excretion of Lead in the Rat. Brit. J. Ind. Med. 21(4):308-314.

Center for Disease Control (Bureau of Epidemiology and National Institute for Occupational Safety and Health). 1977. A Report of Recent Medical Studies of Five U. S. Lead Plants. Bureau of Epidemiology and National Institute for Occupational Safety and Health. Center for Disease Control, Department of Health, Education, Welfare.

Chaube, S., C. A. Swinyard, and H. Nishimura. 1972. A Quantitative Study of Human Embryonic and Fetal Lead with Considerations of Material-Fetal Lead Gradients and the Effect of Lead on Human Reproduction. Teratology 5(2):253.

Chisolm, J. J., Jr., M. B. Barrett, and E. D. Mellits. 1975. Dose-Effect and Dose-Response Relationships for Lead in Children. J. Pediatr. 87: 1152-1160.

Chisolm, J. J. 1977. Is Lead Poisoning Still a Problem? Clin. Chem. 23(2): 252-255.

Clarkson, T. W., and J. E. Kench. 1958. Uptake of Lead by Human Erythocytes *in vitro*. Biochem. J. 69:432-439.

Cooper, W. C. and W. R. Gaffey. 1975. Mortality of Lead Workers. J. Occup. Med. 17(2):100-107.

Coulston, F. 1973. Evaluating Toxicological Data as Regards Environmental Significance. In: EQS Environmental Quality and Safety. Global Aspects of Chemistry, Toxicology and Technology as Applied to the Environment. Vol. 2, F. Coulston and F. Korte, (Eds.). Academic Press, New York. pp. 125-234.

Coulston, F., L. Goldberg, T. B. Griffin, and J. C. Russell. 1972. The Effects of Continuous Exposure to Airborne Lead. Final Report to the U. S. Environmental Protection Agency.

Cramer, K., R. A. Goyer, R. Jagenburg, and M. H. Wilson. 1974. Renal Ultrastructure, Renal Function, and Parameters of Lead Toxicity in Workers with Different Periods of Lead Exposure. Brit. J. Ind. Med. 31(2):113-127.

Cremer, J. E. 1959. Biochemical Studies on the Toxicity of Tetraethyl Lead and Other Organo-Lead Compounds. Brit. J. Ind. Med. 16(3):191-199.

Cremer, J. E., and S. Callaway. 1961. Further Studies on the Toxicity of Some Tetra and Trialkyl Lead Compounds. Brit. J. Ind. Med. 18(4): 277-282.

Daines, R. H., D. W. Smith, A. Feliciano, and J. R. Trout. 1972. Air Levels of Lead Inside and Outside Homes. Indust. Med. Surgery. 41(10): 26-28.

David, O., J. Clark, and K. Voeller, 1972. Lead and Hyperactivity. Lancet. 2:900-903.

Day, J. P., M. Hart, and M. S. Robinson. 1975. Lead in Urban Street Dust. Nature. 253:343-345. January 31.

Deknudt, Gh., A. Leonard, and B. Ivanov. 1973. Chromosome Aberrations Observed in Male Workers Occupationally Exposed to Lead. Environ. Physiol. Biochem. 3(3): 132-138.

de la Burde, B., and M. S. Choate, Jr. 1972. Does Asymptomatic Lead Exposure in Children Have Latent Sequelae? J. Pediat. 81:1086-1091.

de La Burde, B., and M. S. Choate. 1975. Early Asymptomatic Lead Exposure and Development At School Age. J. Pediat. 87:638-642.

Dingwall-Fordyce, J., and R. E. Lane. 1963. A Followup Study of Lead Workers. British J. of Ind. Med. 20:313-315.

Dobbing, J. 1974. The Later Growth of the Brain and Its Vulnerability. Pediatrics. 53:2-6.

Dobbing, J., and J. Sands. 1973. Quantitative Growth and Development of Human Brain. Arch. Dis. Child. 48:756-767.

Eldridge, W. A. 1924. A Study of the Toxicity of Lead Tetraethyl. Chemical Warfare Service. Report E. A. M. R. D. 29 pp.

Ewing, B. B., and J. E. Pearson. 1974. Lead in the Environment. From Advances in Environmental Science and Technology, Vol. 3. J. N. Pitts, Jr., and K. L. Metcalf, Editors. pp. 1-126.

Ferm, V. H. 1969. The Synteratogenic Effect of Lead and Cadmium, Experentia. 25(1):56-57.

Ferm, V. H., and S. J. Carpenter. 1967. Development Malformations Resulting from the administration of Lead Salts, Exp. Mol. Pathol., 7(2):208-213.

Fine, P. R., C. W. Thomas, R. H. Suho, R. E. Cohnberg, and B. S. Flashner. 1972. Pediatric Blood Levels. A Study in 14 Illinois Cities of Intermediate Population. J. Amer. Med. Assoc. 221:1475-1479.

Finklea, J. R., J. C. Creason, V. Hasselblad, D. I. Hammer, L. E. Priester, S. H. Sandifer, and J. E. Keil. 1972. Transplacental Transfer of Toxic Metals. Proc. 17th Int. Cong. Occup. Health, Buenos Aires. pp. 17-23.

Fisher, H. L., Jr. 1969. A Model for Estimating the Inhalation Exposure to Radon-222 and Daughter Products from the Accumulated Lead-210 Body Burden. Health Phys. 16(5):597-616.

Forbes, G. B., and J. C. Reina. 1972. Effect of Age on Gastrointestinal Absorption of Lead in the Rat. J. Nutr. 102:647-652.

Forni, A., and G. C. Secchi. 1973. Chromosome Changes in Preclinical and Clinical Lead Poisoning and Correlation with Biochemiacl Findings in: Proc., Int. Symp., Environ. Health Aspects of Lead, Amsterdam, Oct. 2-6, 1972, D. Barth, A. Berlin, R. Engle, P. Recht, and J. Smeets (eds.), pp. 473-485.

Forni, A., G. Cambiaghi, and G. C. Secchi. 1976. Initial occupational exposure to lead. Chromosome and Biochemical findings. Arch. Environ. Health. 31:73-78.

Fugas, M., B. Wilder, R. Paukovic, J. Hrsak, and D. Steiner-Skreb. 1973. Concentration Levels and Particle Size Distribution of Lead in the Air of an Urban and an Industrial Area as a Basis for the Calculation of Population Exposure. In: Proceedings of the International Symposium; Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972. Luxembourg, Commission of European Communities. pp. 961-968.

Gage, J. C., and M. H. Litchfield. 1969. The Migration of Lead from Paint Films in the Rat Gastro-Intestinal Tract. J. Oil Col. Chem. Assoc. 52:236-243.

Galzigna, L., M. V. Ferraro, G. Manani, and A. Viola. 1973. Biochemical Basis for the Toxic Effects of Triethyl Lead, Brit. J. Ind. Med., 30(2): 129-133.

Gerarde, H. W. 1964. Toxicology: Organic, Ann. Rev. Pharmacol., 4:233-246.

Gibson, S. L. M., J. C. Mackenzie, and A. Goldberg. 1968. The Diagnosis of Industrial Lead Poisoning. Brit. J. Ind. Med. 25(1):40-51.

Gilsinn, J. F. 1972. Estimates of the Nature and Extent of Lead Paint Poisoning in the United States. U. S. Department of Commerce. National Bureau of Standards. Technical Note 746. December.

Goldsmith, J. R., and A. C. Hexter. 1967. Respiratory Exposure to Lead: Epidemilogical and Experimental Dose-Response Relationships. Science. 158(3797):132-134.

Goldwater, L. J. 1972. An Assessment of the Scientific Justification for Establishing 2 μg/m^3 as the Maximum Safe Level for Airborne Lead. Industrial Medicine. 41(7):13-18.

Goldwater, L. J., and A. W. Hoover. 1967. An International Study of "Normal" Levels of Lead in Blood and Urine. Arch. Environ. Health. 15(7):60-63.

Gothe, C. J., H. Ohman, and G. Linstedt. 1973. Exposure to Airborne Lead in City Atmosphere. Work, Environment, and Health. 10:13-18

Goyer, R. A. 1971. Lead Toxicity: A Problem in Environmental Pathology, Am. J. Pathol., 64(1):167-182.

Goyer, R. A., and M. A. Mehlman. 1977. Lead Toxicity Laboratory Aspects. In: Toxicology of Trace Elements. Hemisphere Publishing Corporation. John Wiley and Sons, New York, N. Y. pp. 41-77.

Goyer, R. A., and B. C. Rhyne. 1973. Pathological Effects of Lead. Int. Rev. Exp. Pathol. 12:1-77.

Griffin, T. B., F. Coulston, H. Wills, J. C. Russell, J. H. Knelson. 1975. Clinical Studies on Men Continuously Exposed to Airborne Particulate Lead. In: Environmental Quality and Safety, Suppl. Vol. II. Georg. Thieme, Stuttgart.

Gross, S. B., and E. A. Pfitzer, 1975. Influence of Pathological Changes in Lead in Human Tissues. Trace Substances in Environmental Health - III, Proc. 8th Ann. Conf. on Trace Substances in Environ. Health, University of Missouri, Columbis, MO, June 11-14, 1974, D. D. Hemphill, Editor.

Gross, S. B., E. A. Pfitzer, D. W. Yeager, and R. A. Kehoe. 1975. Lead in Human Tissues. Toxicol. Appl. Pharmacol. 32(3):638-651.

Gruden, N., and M. Stantic. 1975. Transfer of Lead Through the Rat's Intestinal Wall. Sci. Total Environ. 3(3):288-292.

Haas, T., K. Mache, K. Schaller, A. Wieck, W. Mache, and H. Valentin. 1973. Undersuchugen uber die Okologische Bleibelastung in Kindesalter. Proc., Int. Symp., Environ. Health Aspects of Lead, Amsterdam, Oct. 2-6, 1972. D. Barth, A. Berlin, R. Engel, P. Recht, and J. Smeets, Editors. pp. 741-748.

Haeger-Aronsen, B. 1971. An Assessment of the Laboratory Tests Used to Monitor the Exposure of Lead Workers. Brit. J. of Ind. Med. 28( ):52-58.

Hamilton, A., and H. L. Hardy, 1974. Industrial Toxicology, 3rd Edition, Publishing Sciences Group, Inc., Anton, Mass. 575 pp.

Hammond, P. B. 1977, University of Cincinnati. Personal Communication with Mary Anne Bell, Battelle Columbus Laboratories. Informal memo titled "Appendix A, ... the validity of a mathematical model describing the relationship of air lead to concentration of lead in blood using model developed by Azar, et al., 1973".

Hammond, P. B. 1978. Environmental Assessment. In: Review of the Environmental effects of Pollutants: VII. Lead. EPA-600/1-78-029. U. S. Environmental Protection Agency, Health Effects Research Laboratory, Cincinnati, Ohio Various pagination.

Hammond, P. B., and A. L. Aronson. 1964. Lead Poisoning in Cattle and Horses in the Vicinity of a Smelter. Ann. N. Y. Acad. Sci. III, Art. 2. pp. 595-611.

Harris, P., and M. R. Holley. 1972. Lead Levels in Cord Blood, Pediat. 49(4): 606-608.

Hertzig, M. E., H. G. Birch, S. A. Richardson, and J. Tezard. 1972. Intellectual Levels of School Children Severely Malnourished During the First Two Years of Life. Pediat. 49:814-824.

Hilderbrand, D., M. Olds, R. Der, and M. S. Fahim. 1973. Effect of Lead Acetate on Reproduction and Metabolism. Trace Substances in Environmental Health - VI, 6th Ann. Conf. on Trace Substances in Environmental Health, Univ. of Missouri, Columbia, Mo., June 13-15, 1972. D. D. Hemphill, (Ed.). 237-246.

Hine, C. H., R. D. Cavalli, and S. M. Beltram. 1969. Percutaneous Absorption of Lead from Industrial Lubricants. J. Occup. Med. 11:568-575.

Hofreuter, D. H., E. J. Catcott, R. G. Keenan, and C. Xintaras. 1961. The Public Health Significance of Atmospheric Lead. Arch. Environ. Health 3(11):568-574.

Holtzman, R. B. 1963. Measurement of the Natural Contents of RaD (Pb210) in Human Bone - Estimates of Whole-Body Burdens. Health Phys. 9(4):385-400.

Horiuchi, K. 1970. Lead in the Environment and Its Effect on Man in Japan. Osaka City Med. J. 16:1-28.

Horiuchi, K., S. Horiguchi, and M. Suekane. 1959. Studies on the Industrial Lead Poisoning. I. Absorption, Transportation, Deposition and Excretion of Lead. 6. The Lead Contents in Organ-Tissues of the Normal Japanese. Osaka City Med. J. 5(1):41-70.

Horiuchi, K., and I. Takada. 1954. Studies on the Industrial Lead Poisoning. I. Absorption, Transportation, Deposition, and Excretion of Lead. 2. Normal Limits of Lead in the Blood, Urine, and Feces Among Healthy Japanese Urban Habitants. Osaka City Med. J. 1:117-125.

Hursh, J. B. 1973. Retention of 210 Pb in Beagle Dogs, Health Phys., 25:29-35.

ICRP, 1959. Recommendations of the International Commission on Radiological Protection, Report of Committee II on Permissible Dose for Internal Radiation, Publication No. 2, Pergamon Press, London.

Iwata, H., M. Kawai, Y. Katagiri, and M. Hirsta. 1971. Effects of Lead in Air During Work on Traffic Controllers. Figu Daigaku Izakubu Kiyo 18(5): 479-485.

Jowett, M. 1932. Reactions of Lead Compounds with Serum and Serum Models. Biochem. J. 26:2108-2122.

Jung, F. 1947. Pathology of the Red Blood Cells. II. Action of Some Metallic Salts. Arch. Exp. Pathol. Pharmakol. 204:138-156.

Kammholz, L. P., L. G. Thatcher, F. M. Blodgett, and T. A. Good. 1972. Rapid Protoporphyrin Quantitation for Detection of Lead Poisoning. Pediat. 50:625-631.

Karlag, O., and K. O. Moeller. 1958. Three Cases of Acute Lead Poisoning. Analyses of Organs for Lead, and Observations on Polarographic Lead Determinations. Acta. Pharmacol. Toxicol. 15(1):8-16.

Kehoe, R. A. 1925. Tetraethyl Lead Poisoning: Clinical Analysis of a Series of Nonfatal Cases. J. Am. Med. Ass. 85:108-110.

Kehoe, R. A. 1927. On the Toxicity of Tetraethyl Lead and Inorganic Lead Salts. J. Lab. Clin. Med. 12:554-560.

Kehoe, R. A., and F. Thamann. 1931. The Behavior of Lead in the Animal Organism. II. Tetraethyl Lead. Am J. Hyg. 13(3):478-498.

Kehoe, R. A. 1961a. The Harben Lectures, 1960: The Metabolism of Lead in Man in Health and Disease. 1. The Normal Metabolism of Lead. J. Roy. Inst. Publ. Health Hyg. 24:81-97.

Kehoe, R. A. 1961b. The Harben Lectures, 1960: The Metabolism of Lead in Man in Health and Disease. 2. Part 1. The Metabolism of Lead Under Abnormal Conditions. J. Roy. Inst. Publ. Health Hyg. 24:101-121.

Kehoe, R. A. 1961c. The Harben Lectures, 1960: The Metabolism of Lead in Man in Health and Disease. 2. Part 2. The Metabolism of Lead Under Abnormal Conditions. J. Roy. Inst. Publ. Health Hyg. 24:129-143.

Kehoe, R. A. 1961d. The Harben Lectures, 1960: The Metabolism of Lead in Man in Health and Disease. 3. Present Hygienic Problems Relating to the Absorption of Lead. J. Roy. Inst. Publ. Health Hyg. 24:177-203.

Kehoe, R. A. 1964. Metabolism of Lead Under Abnormal Conditions. Arch. Environ. Health. 8(2):235-243.

Kehoe, R. A. 1966. Data from Four Subjects Experimentally Exposed to Lead Through Inhalation. Proceedings of Fifteenth Congress Intern. Med. Travail., Vienna. 3:83.

King, B. G., and A. F. Schaplowsky. 1974. Basic Factors in Evaluation of Hazard of Childhood Lead Exposures. Presented at the Annual Meeting of the American Public Health Assoc., New Orleans, La., October 23.

Klein, P. S., G. B. Forbes, and P. R. Nader. 1975. Effects of Starvation in Infancy (Pyroloric Stenosis) on Subsequent Learning Abilities. J. Pediatr. 87:8-15.

Knelson, J. H., R. J. Johnson, F. Coulston, L. Goldberg, and T. Griffin. 1973. Kinetics of Respiratory Lead Uptake in Humans. Proc. Int. Symp., Environ. Health Aspects of Lead. Amsterdam, October 2-5, 1972, D. Barth, A. Berlin, R. Engel, P. Recht, and J. Smeets (Eds.), pp. 391-401.

Kolbye, A. C., Jr., K. R. Mahaffey, J. A. Fiorino, P. C. Corneliussen, and C. F. Jelinek. 1974. Food Exposures to Lead. Environ. Health Perspect., Exptl. Issue No. 7:65-74. May.

Kostial, K., and B. Momcilovic. 1974. Transport of Lead 203 and Calcium 47 from Mother to Offspring. Arch. Environ. Health 29(1):28-30.

Kostial, K., I. Simonovic, and M. Pisonic. 1971a. Reduction of Lead Absorption from the Intestine in Newborn Rats. Environ. Res. 4(4):360-363.

Kostial, K., I. Simonovic, and M. Pisonic. 1971b. Lead Absorption from the Intestine in Newborn Rats. Nature 233(5321):546.

Kruckenberg, S. M., S. M. Dennis, H. W. Leipold, F. W. Oehme, and J. E. Cook. 1976. Teratogenicity of Lead Acetate in Microtus Ochrogester (Prarie Voles). Vet. Toxicol. 18(2):58-60.

Kubota, J., V. A. Lazar, and F. Losee. 1968. Copper, Zinc, Cadmium, and Lead in Human Blood from 19 Locations in the United States. Arch. Environ. Health 16(6):788-793.

Lancranjan, J., H. I. Popescu, O. Gavanescu, I. Klepsch, and M. Serbanescu. 1975. Reproductive Ability of Workmen Occupationally Exposed to Lead, Arch. Environ. Health, 30(8):396-401.

Lamola, A. A., and Yamane, T. Zinc Protoporphyrin in the Erythrocytes of Patients with Lead Intoxication and Iron Deficiency Anemia. Science 186:936-938, 1974.

Landrigan, P. J., S. H. Gehlback, B. F. Rosenblum, J. M. Shoults. 1975a. Epidemic Lead Absorption Near an Ore Smelter: The Role of Particulate Lead, New England J. of Med., 292(3):123-124.

Landrigan, P. J., W. F. Whitworth, R. W. Balch, N. W. Staehling, W. F. Barthel, and B. F. Rosenblum. 1975b. Neuropsychological Dysfunction in Children with Chronic Low-Level Lead Absorption. Lancet. 1:708-712.

Landrigan, P. J., A. S. McKinney, L. C. Hopkins, W. W. Rhodes, Jr., W. A. Price and D. H. Cox. 1975c. Chronic Lead Absorption, Results of Poor Ventilation in an Indoor Pistol Range, J. of the Amer. Med. Assoc., 234(4): 394-397.

Landrigan, P. J., E. L. Baker, Jr., R. G. Feldman, D. H. Cox, K. V. Eden, W. A. Orinsken, J. A. Mather, A. J. Yankel, and I. H. von Lindern. 1976. Increased Lead Absorption with Anemia and Slower Nerve Conduction in Children Near a Lead Smelter. J. Pediatr. 89(6):904-910. December.

Lansdown, R. G., P. J. Shepherd, B. E. Clayton, H. T. Delves, P. J. Graham, and W. C. Turner. 1974. Bloodlead Levels, Behavior, and Intelligence; A Population Study. Lancet. 1:538-541.

Laug, E. P., and F. M. Kunze. 1948. The Penetration of Lead Through the Skin, J. Ind. Hyg. Toxicol., 30:256-259.

Lawther, P. J., B. T. Cummins, J. McEllison and B. Biles. 1972. Airborne Lead and Its Uptake by Inhalation. In: Lead in the Environment, Proc. Conf. Zool. Soc. London, Regents Park, P. Hepple, Editor, pp. 8-28.

Lepow, M. L., L. Bruckman, R. A. Rubino, S. Markowitz, M. Gillette, and J. Kopish. 1974. Role of Airborne Lead in Increased Body Burden of Lead in Hartford Children. Environ. Health Perspect., Exptl. Issue No. 7:99-102, May.

Lepow, M. L., L. Bruckman, M. Gillette, S. Markowitz, R. Robins, and J. Kapish. 1975. Investigations into Sources of Lead in Environment of Urban Children. Env. Res. 10:415-426.

Malcolm, D. 1971. Prevention of Long-Term Sequellae Following the Absorption of Lead. Arch. Environ. Health, 23:123-124.

Martell, E. A. 1975. Tobacco Radioactivity and Cancer in Smokers, Am. Sci. 63:404-412.

Martin, A. E. 1974. Epidemiological Surveys in the Vicinity of Lead Works. Proc. Roy. Soc. Med. 67:165-167.

Maxwell, L. C., and F. Bischoff. 1929a. The Reaction of Lead with the Constituents of the Erythrocytes. J. Pharmacol. Exp. Ther. 37(4): 413-428.

Maxwell, L. C., and F. Bischoff. 1929b. The Solubility of Lead Salts in Physiological Salt Solutions. J. Pharmacol. Exp. Ther. 36(3):279-293.

McClain, R. M., and B. A. Becker. 1972. Effects of Organolead Compounds on Rat Embryonic and Fetal Development. Toxicol. Appl. Pharmacol., 21(2):254-274.

McIntyre, M. S. and C. R. Angle. 1972. Air Lead: Relation to Lead in Blood of Black School Children Deficient in Glucose - 6 - Phosphate Dehydrogenase. Science. 177(4048):520-522.

Mehani, S. 1966. Lead Retention by the Lungs of Lead-Exposed Workers. Ann. Occup. Hyg. 9(3):165-171.

Millar, J. A., G. G. Thompson, A. Goldberg, P. S. I. Barry, and E. H. Lowe. 1972. Aminolevulinic Acid Dehydrase Activity in the Blood of Men working with Lead Alkyls. Brit. J. Ind. Med. 29(3):317-320.

Momcilovic and K. Kostial. 1974. Kinetics of Lead Retention and Distribution in Suckling and Adult Rats. Environ. Res. 8:214-220.

Morris, H. P. 1940. Age and the Lead Content of Certain Human Bones: A Compilation and Statistical Analysis of Recently Published Data. J. Ind. Hyg. Toxicol. 22(3):100-103.

Mortensen, R. A. 1942. The Absorption of Lead Tetraethyl with Radioactive Lead as Indicator. J. Ind. Hyg. Toxicol. 24(9):285-287.

Mortensen, R. A., and K. E. Kellogg. 1944. The Uptake of Lead by Blood Cells as Measured with a Radioactive Isotope. J. Cell. Comp. Physiol. 23(1): 11-20.

Muro, L. A., and R. A. Goyer. 1969. Chromosome Damage in Experimental Lead Poisoning. Arch. Pathol. 87:660-663.

National Academy of Sciences, Committee on Biologic Effects of Atmospheric Pollutants. 1972. Biologic Effects of Atmospheric Pollutants. Lead. Airborne Lead in Perspective, Washington, D. C., 341 pp.

National Academy of Sciences. 1976. Recommendations for the Prevention os Lead Poisoning in Children. Committee on Toxicology. Washington, D. C.

Neal, P. A., W. C. Dressen, T. J. Edwards, W. H. Reinhart, S. H. Webster, H. T. Castberg and L. T. Fairhall. 1941. A Study of the Effect of Lead Arsenate Exposure on Archardists and Consumers of Sprayed Fruit, U. S. Public Health Bulletin #267. U. S. Government Printing Office, Washington, D. C.

Nelson, W. C., M. H. Lykins, J. Mackey, W. A. Newill, J. F. Finklea, and D. I. Hammer. 1973. Mortality Among Orchard Workers Exposed to Lead Arsenate Spray; a Cohort Study. J. Chron. Dis. 26:105-118.

Nordman, C. H., S. Hernberg, J. Nikkanen, and A. Ryhanen. 1973. Blood Lead Levels and Erythrocyte delta - Aminolevulinic Acid Dehydratase Activity Living Around a Secondary Lead Smelter. Work Environ. Health. 10(1): 19-25.

Nozaki, K. 1966. Method for Studies on Inhaled Particles in Human Respiratory System and Retention of Lead Fume, Industrial Health (Japan). 4(X):118-128.

O'Riardan, M. L., and H. J. Evans. 1974. Absence of Signigicant Chromosome Damage in Males Occupationally Exposed to Lead. Nature. 274:50-53

Oyasu, R., H. A. Battifora, R. A. Clasen, J. H. McDonald, and G. M. Haas. 1970. Induction of Cerebral Gliomas in Rats with Dietary Lead Subacetate and 2-Acetylaminoflourene. Cancer Res. 30:1248-1261.

Palmisano, P. A., R. C. Sneed, and G. Cassady. 1969. Untaxed Whiskey and Fetal Lead Exposure. J. Pediat. 75(5):869-872.

Passow, H., and K. Tillmann. 1955. Potassium Loss from Lead-Poisoned, Human Erythrocytes. Pfluger's Arch. Ges. Physiol. 262:23-36.

Pentschew, A. 1958. Nonallergic Sensitization of the Brain to Lead and Its Significance for the Occurrence and Course of Lead Encephalopathy. Proc. 2nd Intern. Cong. Neuropathol., London, 1955, pp. 569-71.

Pentschew, A., and F. Garro. 1966. Lead Encephalo-Myelopathy of the Suckling Rat and Its Implications on the Porphyrinopathic Nervous Diseases; with Special References to the Permeability Disor-ers of the Nervous System's Capillaries. Acta Neuropathol. 6:266-278.

Perino, J., and C. B. Ernhart. 1974. The Relation of Subclinical Lead Level to Cognitive and Sensorimotor Impairment in Black Preschoolers. J. Learn. Disabil. 7:616-620.

Pettinati, L., L. Rosetti, and G. Rubino. 1959. Intossicazione da Naftenato di Piombo Contenout Negli Olii Lubrificanti per Sistemi Idropneumatici (Poisoning from Lead Naphthenate Present in the Lubricant Oils for Hydropneumatic Systems). Rass . Med. Ind. Ig. Lav. 28(5):379-385.

Piomelli, S., B. Davidow, V. Guinee. 1973. The FEP (Free Erythrocyte Protoporphyrin) Test. Pediatrics. 51:254-257.

Poklis, A. 1975. The Lead Content of the Aorta in Male Residents of Baltimore, Maryland. Bull. Environ. Contam. Toxicol. 13(4):417-419.

Polkis, A., and H. C. Freimuth. 1975. The Lead Content of the Trachea in Baltimore Residents. Bull. Environ. Contam. Toxicol. 14(2):168-170.

Prpic-Majic, D., P. K. Mueller, V. C. Lew and S. Twiss. 1973. Delta Aminolevulinic Acid Dehydratase Stability in Human Blood. Amer. Indust. Hygiene Assoc. J. 315-319.

Pueschel, S. M. 1974. Neurological and Psychomotor Functions in Children with an Increased Lead Burden. Environ. Health Perspect. 7:13-16.

Pueschel, S. M., L. Kopito, and H. Schwachman. 1972. Children with an Increased Lead Burden. A Screening and Follow-Up Study, J. Am. Med. Ass. 222(4):462-466.

Rabinowitz, M. B. 1974. Lead Contamination of the Biosphere by Human Activity; A Stable Isotope Study, Ph.D. Thesis, University of California, Los Angeles, 120 pp.

Rabinowitz, M. B., G. W. Wetherill, and J. D. Kopple. 1974. Studies of Human Lead Metabolism by Use of Stable Isotope Tracers. Environ. Health Perspect. 7:145-153.

Rabinowitz, M., G. Wetherill, and J. Kopple. 1975. Human Uptake of Dietary and Atmospheric Lead: Stable Isotope and Balance Study, In: Trace Contaminants in the Environment, Proc., 2nd Ann. NSF-RANN Trace Contam. Conf., Asilomar, Pacific Grove, Calif., Aug. 29-31, 1974, T. Novakov (chm.), pp. 308-315.

Rajegowda, B. K., L. Glass, and H. E. Evans. 1972. Lead Concentrations in the Newborn Infant. J. Pediat. 80:116-117.

Rastogi, S. C., and J. Clausen. 1976. Absorption of Lead through the Skin. Toxicol. 6(3):371-376.

Reddi, K. K. 1953. Isolation of Thorium B (Lead)-Binding Substance from the Erythrocytes of Rabbit Blood. Nature. 172(4370):202.

Repko, J. D., B. B. Morgan, Jr. and J. Nicholson. 1975. Behavioral Effects of Occupational Exposure to Lead, Division of Laboratories and Criteria Development. National Institute of Occupational Safety and Health, U. S. Department of Health, Education and Welfare, Cincinnati, Ohio, HEW Publication No.75-164, 239 pp.

Roberts, T. M., T. C. Hutchinson, J. Paciga, A. Chattopadhyay, R. E. Jervis, J. Van Loon and D. K. Parkinson. 1974. Lead Contamination Around Secondary Smelters: Estimation of Dispersal and Accumulation by Humans. Science. 186(4169):1120-1123.

Robinson, M. J., F. E. Karpinski, Jr., and H. Brieger. 1958. The Concentration of Lead in Plasma, Whole Blood and Erythrocytes of Infants and Children Pediat. 21:793-797.

Robinson, T. R. 1974. Delta-Aminolevulinic Acid and Lead in Urine of Lead Antiknock Workers. Arch. Environ. Health. 28(3):133-136.

Robinson, T. R. 1976. The Health of Long Service Tetraethyl Lead Workers. J. Occup. Med. 18(1):31-40.

Rosen, J. F., and E. E. Trinidad. 1974. Significance of Plasma Lead Levels in Normal and Lead-Intoxicated Children. Environ. Health Perspect., Exptl. Issue No. 7:139-144, May.

Rosen, J. F., C. Zarate-Salvadore, and E. E. Trinidad. 1974. Plasma Lead Levels in Normal and Lead-Intoxicated Children. J. Pediatr. 84(1):45-48.

Rosetti, L., L. Pettinati and G. Scansetti. 1961. Chronic Lead Naphthenate Poisoning, Transcultaneous Absorption of the Compound, Proceedings of the 13th International Congress for Occupational Health, Waldorf - Astoria Hotel, New York, July 25-29, 1960, pp. 384-389.

Sanders, L. W., Sr. 1964. Tetraethyllead Intoxication. Arch. Environ. Health. 8(2):270-277.

Sassa, S., J. L. Granick, S. Granick, A. Kappas, and R. D. Levere. 1973. Studies in Lead Poisoning. I. Microanalysis of Erythrocyte Protoporphyrin Levels by Spectrofluorometry in the Detection of Chronic Lead Intoxication in the Subclinical Range. Biochem. Med. 8:135-148.

Sayre, J. W. E. Charney, J. Vostal, and I. B. Pless. 1974. House and Hand Dust as a Potential Source of Childhood Lead Exposure. Am. J. Dis. Child. 127:167-170.

Scanlon, J. 1971. Umbilical Cord Blood Lead Concentration: Relationship to Urban or Suburban Residency During Gestation. Am. J. Dis. Child. 121(4): 325-326.

Schepers, G. W. H. 1964. Tetraethyllead and Tetramethyllead, Comparative Experimental Pathology: Part I. Lead Absorption and Pathology. Arch. Environ. Health. 8(2):277-295.

Schroeder, H. A. 1973. Recondite Toxicity of Trace Elements, In: Essays in Toxicology. Vol. 4, W. J. Hayes (Ed.), Academic Press, New York, pp. 107-199.

Schmid, E., M. Bauchinger, S. Pietruck and G. Hall. 1972. Diecytogenetische Wirkung von Blei in menschlichen peripheren Lymphocyten *in vitro* und *in vivo*. *Mutat. Res.* 16:401-406.

Schroeder, H. A., and J. J. Bala-sa, 1961. Abnormal Trace Metals in Man: Lead. J. Chron. Dis. 14(4):408-425.

Schroeder, H. A., and M. Mitchener. 1971. Toxic Effects of Trace Elements on the Reproduction of Mice and Rats. Arch. Environ. Health. 23(2): 102-106.

Schroeder, H. A., and I. H. Tipton. 1968. The Human Body Burden of Lead. Arch. Environ. Health. 17(12):965-978.

Schwanitz, G., E. Gebhart, H. D. Rott, K. H. Schaller, H. G. Essing, O. Lauer, O., and H. Prestele. 1975. Chromosome investigations in subjects with occupational lead exposure. *Disch. Med. Wschr.* 100:1007-1011.

Schwanitz, G., G. Lehnert, and E. Gebhart. 1970. Chromosomenschäden bei beruflicher Bleibelastung. *Disch. Med. Wschr.* 95:1636-1641.

Selander, S. and K. Cramer. 1970. Interrelationships Between Lead in Blood, Lead in Urine and Ala in Urine During Lead Work. Brit. J. Ind. Med. 27:28-39.

Seppalainen, A. M., S. Tola, S. Heinberg and B. Kock. 1975. Subclinical Neuropathy at Safe Levels of Lead Exposure. Arch. of Environ. Health, 30(4):180-183.

Shapiro, H., and F. W. Frey. 1968. The Organic Compounds of Lead. Interscience Publishers, New York, 486 pp.

Short Associates. 1976. Preliminary Technological Feasibility, Cost of Compliance, and Economic Impact Analysis of the Proposed OSHA Standard for Lead. Prepared for OSHA. U. S. Department of Labor, by J. Short and Associates, Salt Lake City, Utah. 327 pp.

Six, K. M., and R. A. Goyer. 1972. The Influence of Iron Deficiency on Tissue Content and Toxicity of Ingested Lead in the Rat. J. Lab. Clin. Med. 79:128-136.

Sobel, A. E., O. Gawron, and B. Kramer. 1938. Influence of Vitamin D. in Experimental Lead Poisoning. Proc. Soc. Exp. Biol. Med. 38(3):433-435.

Stevens, C. D., C. J. Feldhake, and R. A. Kehoe. 1956. Isolation from Liver of Tetraethyllead After Its Inhalation. J. Pharmacol. Exp. Ther. 117(4): 420-424.

Stover, B. J. 1959. Pb^{212}(ThB) Tracer Studies in Adult Beagle Dogs. Proc. Soc. Exp. Biol. Med. 100(2):269-272.

Stowe, H. D., and R. A. Goyer. 1971. Reproductive Ability and Progemy of F_1 Lead-Toxic Rats. Fertil. Steril. 22:755-760.

Sumino, K., K. Hayakawa, T. Shibata, and S. Kitamura. 1975. Heavy Metals in Normal Japanese Tissues. Amounts of 15 Heavy Metals in 30 Subjects. Arch. Environ. Health. 30(10):487-494.

Sunderman, F. W., Jr. 1971. Metal Carcinogenesis in Experimental Animals. Fd. Cosmet. Toxicol. 9(1):105-120.

Task Group on Metal Accumulation (TGOMA). 1973. Accumulation of Toxic Metals with Special Reference to Their Absorption, Excretion and Biological Half-Times. Environ. Physiol. Biochem. 3(2):65-107.

Teisinger, J., R. Zumanova, and I. Zezula. 1958. Effect of Edathamil Calcium-Disodium on the Lead Content of Red Blood Cells and Blood Protiens. Arch. Ind. Health. 17(4):295-301.

Tepper, L. B. and L. S. Levin. 1975. A Survey of Air and Population Lead Levels in Selected American Communities. In: Lead. T. B. Griffin and J. H. Knelsor (eds.). Environmental Quality and Safety. Supplement Vol. II. Georg. Thieme Publishers, Stuttgart. pp 152-196.

Ter Haar, G. L., and R. Aronow. 1974. New Information on Lead in Diet and Dust as Related to the Childhood Lead Problem, Environ. Health Perspect. Experimental Issue (7):83-89. May.

Texas Morbidity and Mortality Weekly Report. 1973. Human Lead Absorption, Texas Morbidity and Mortality Weekly Report. 22(49):405-407. December 8.

Thomas, H. V., B. K. Milmore, G. A. Heidbreder and B. A. Kogan. 1967. Blood Lead of Persons Living Near Freeways. Arch. of Environ. Health. 15(6): 695-705.

Tipton, I. H., and M. J. Cook. 1963. Trace Elements in Human Tissue. Part II. Adult Subjects from the United States. Health Phys. 9(2):103-145.

Tipton, I. H., H. A. Schroeder, H. M. Perry, Jr., and M. J. Cook. 1965. Trace Elements in Human Tissue. Part III. Subjects from Africa, The Near and Far East and Europe. Health Phys. 11(5):403-451.

Tipton, I. H., and J. J. Shafer. 1964. Statistical Analysis of Lung Trace Element Levels. Arch. Environ. Health. 8(1):58-67.

Tola, S., S. Hernberg and J. Nikkanen. 1972. Occupational Lead Exposure in Finland. II Service Stations and Garages, Work, Environment, Health. 9: 102-105.

Tola, S., S. Hernberg, J. Nikkanen and S. Valkonen. 1973. Parameters Indicative of Absorption and Biological Effect in New Lead Exposure, Brit. J. of Indust. Med. 30:134-141.

Tolgskaya, M. S. and A. B. Reznikov. 1954. Morphologic Changes of the Central Nervous System in Tetraethyl Lead Poisoning. Trudy Akademii Meditsinskikn Nauk, U.S.S.R. 31:168-189.

Torvik, E., E. Pfitzer, J. B. Kereiakes, and R. Blanchard. 1974. Long Term Effective Half-Lives for Lead 210 and Polonium-210 in Selected Organs of the Male Rat. Health Phys. 26(1):81-87.

U. S. Department of Health, Education, and Welfare, Working Group on Lead Contamination. 1965. Survey of Lead in the Atomsphere of Three Urban Communities, Division of Air Pollution, Public Health Service, U. S. Dept. of Health, Education and Welfare, Cincinnati, Ohio PHS Publication No. 999-AP-12. 94 pp.

U. S. Department of Health, Education, and Welfare. 1972. Ten-State Nutrition Survey, 1968-1970. V. Dietary. Center for Disease Control, Atlanta, Georgia. DHEW Pub. No. (HSM)72-8133.

U. S. Department of Labor. 1975. Occupational Exposure: Proposed Standard, Occupational Safety and Health Administration. 40 FR 45934-45948, October 3.

U. S. Department of Labor. 1978. Occupational Exposure to Lead: Final Standard. Occupational Safety and Health Administration, Washington, D.C. 43 FR 52952-53014, November 14.

U. S. Environmental Protection Agency. 1972. EPA's Position on the Health Effects of Airborne Lead. Health Effects Branch, Processes and Effects Division, Office of Research and Monitoring. Washington D. C. 106 pp.

U. S. Environmental Protection Agency. 1976. Environmental Assessment of the Domestic Primary Copper, Lead and Zinc Industries, Volume II - Appendices, U. S. Environmental Protection Agency, Industrial Environmental Research Laboratory, Cincinnati, Ohio.

U. S. Environmental Protection Agency. 1977. Air Quality Criteria for Lead. EPA-600/3-77-017. Ofice of Research and Development, Washington, D.C. December. Various Pagination.

U. S. Environmental Protection Agency. 1978. National Primary and Secondary Air Quality Standard for Lead. 43 FR 46246-46263. October 5.

Vallee, B. L., and D. D. Ulmer. 1972. Biochemical Effects of Mercury, Cadmium, and Lead. Ann. Rev. Biochem. 41:91-128.

van Peteghem, Th., and H. de Vos. 1974. Toxicity Study of Lead Naphthenate. Brit. J. Ind. Med. 31(3):233-238.

Vermande - Van Eck, G. J., and J. W. Meigs. 1960. Changes in the Ovary of the Rhesus Monkey After Chronic Lead Intoxication. Fertil, Steril. 11: 223-234.

Vincent, P. C. 1958. The Effects of Heavy Metal Ions on the Human Erythrocyte. II. The Effects of Lead and Mercury. Aust. J. Exp. Biol. Med. Sci. 36(6): 589-601.

Vitale, L. F., M. M. Joselowk, R. P. Wedeen and M. Pawlow. 1975. Blood-Lead - An Inadequate Measure of Occupational Exposure J. Occup. Med. 17(3): 155-156.

von Lindern, I. and A. J. Yankel. 1976. Presentation to the Shoshone Heavy Metals Project Committee. *In*: Shoshone Lead Health Project Work Summary. January.

Vostal, J. 1963. Mechanisms of Lead Excretion. Biochem. Pharmacol. 12:207.

Vostal, J. 1966. Study of the Renal Excretory Mechanism of Heavy Metals. Proc. 15th Int. Cong. Occup. Health. Vienna, September 19-24, 1966, Vol. 3, 61 pp.

Vostal, J., and J. Heller. 1968. Renal Excretory Mechanisms of Heavy Metals. 1. Transtubular Transport of Heavy Metal Ions in the Avian Kidney. Environ. Res. 2(1):1-10.

Waldron, H. A., and D. Stofen. 1974. Sub-Clinical Lead Poisoning. Academic Press, New York. 224 pp.

Wall Street Journal. 1977a. U. S. Safety Aides Link Buildup of Lead in Worker's Blood to Exposure by Air - By Gail Bronson, Staff Reporter, Wall Street Journal, Tuesday, March 15.

Wall Street Journal. 1977b. Study Shows Children of Lead Workers are Susceptible to Lead-Dust Poisoning, Wall Street Journal. February 3.

Williams, M. K. 1972. Criteria for a Recommended Standard Occupational Exposure to Inorganic Lead, as Cited on pp V-9, U. S. Department of Health, Education and Welfare, Public Health Service, National Institute for Occupational Safety and Health.

Williams, M. K. 1975. Justification for NIOSH Recommended Air Standard. J. Occup. Med. 17(2):93-96.

Williams, M. K., E. King and J. Walford. 1969. An Investigation of Lead Absorption in an Electric Accumulator Factory with the Use of Personal Samplers. Brit. J. Indust. Med. 26:202-216.

World Health Organization. 1977. Environmental Health Criteria, 3. Lead. Geneva, United States Environmental Programme and the World Health Organization. 133 pp.

Yankel, A. J., I. von Lindern, and S. D. Walter. 1977. The Silver Valley Lead Study. The Relationship Between Childhood Blood Lead Levels and Environmental Exposure. J. Air Pollut. Cont. Assoc. 27:763-767.

Ziegler, E. E., B. B. Edwards, R. L. Jenson, K. R. Mohaffey, and S. J. Fomon. 1978. Absorption and Retention of Lead By Infants. Ped. Res. 12:29-34.

Zielhuis, R. L. 1975a. Dose-Response Relationships for Inorganic Lead, I. Biochemical and Haematological Responses. Int. Arch. Occup. Health. 35: 1-18.

Zielhuis, R. L. 1975b. Dose-Response Relationships for Inorganic Lead, II. Subjective and Functional Responses - Chronic Sequelae - No Response Levels. Int. Arch. Occup. Health. 35:19-35.

8.0 LEAD IN THE ENVIRONMENT

8.1 SUMMARY

It is the intention of this section to evaluate environmental concentrations of lead in air, water, and food, and the relative contributions from each of these media to total lead absorption and to blood lead levels. Also evaluated is the effect of reducing adventitious lead in foods upon dietary lead intake and, for a selected population sub-group, the effect upon mean blood-lead levels. Much of the discussion centers upon adults in the normal, nonoccupationally exposed general population, for whom the most data are available. The exposure modes and responses of children are more complex and difficult to quantify than those of adults and also critical aspects of the metabolic process, (particularly absorption/retention rates) for various modes of exposure have not been adequately characterized in children as yet. Where it is known that children differ from adults, separate estimates will be presented, if possible. It should be understood that such estimates do not apply to children with pica or other unquantifiable or unusual sources of lead intake.

Humans are exposed to environmental lead through inhalation, ingestion, and in the case of organolead compounds, through cutaneous absorption. Under normal conditions, the concentration of organic lead present in the environment is so low that cutaneous absorption of organic lead can be ignored, except in accidental or occupational exposure cases (U. S. Environmental Protection Agency, 1977). Inhalation is of undisputed importance as the primary contributor to body burden among those occupationally exposed to lead. Furthermore, relative to dietary lead, inhalation may provide an equal or more important route of exposure for persons living in the immediate vicinity of major stationary sources or heavily traveled automobile freeways. These are special exposure conditions which are not applicable to the general adult population, however. Exposure patterns unique to children are discussed in Section 8.6.5.

For the general population, therefore, ingestion appears to be the most important mechanism of exposure. Except in the special circumstances outlined above, exposure via inhalation of ambient air by the general population is clearly of secondary importance compared to exposure via ingestion of food, water and cigarette use.

As indicated in Section 7.5.1, efforts to determine the absorption of lead by the respiratory system at specified air lead concentrations, have been only marginally successful for various technical reasons. It has been necessary to resort to blood lead PbB as an indirect measure of "dose" of air lead. For people who are breathing fairly constant air lead concentrations, averaged over months, the concentration of lead in the blood varies in accordance with the air lead concentration. Analysis of data as to air lead vs. blood lead levels in several studies of adults, indicate that over the range of general ambient air lead concentrations, 1 μg Pb/m^3 of air contributes 1-2 μg Pb/dl in blood. This relationship seems also to apply to children. (Hammond, 1978).

Lead in the atmosphere, resulting, primarily, from the 150,000$^+$ metric tons of lead consumed annually in gasoline antiknock additives, is one significant contributor to human intake and body burdens. Since concentrations are directly related to gasoline consumption, it is not surprising that concentration gradients are seen in the vicinity of heavily traveled highways. Levels above 10 μg/m^3 have been measured in heavy traffic. These concentrations diminish rapidly, almost exponentially, with increasing distance from the highway and atmospheric lead concentrations stabilize at low levels (1-2 μg/m^3), approximately, 100 feet from the road due to the settling of particles (Ewing and Pearson, 1974). Risks to health from airborne ambient lead occur, primarily, among persons living in areas immediately adjacent to freeways and heavily-trafficked urban streets (elevated blood lead seen) and among traffic policemen, auto mechanics, and others who work in an atmosphere of heavy automotive usage with poor ventilation. Persons residing in areas near lead-emitting industries also show elevated blood levels which are indicative of increased absorption of lead (U. S. Environmental Protection Agency, 1977).

However, improvements in air lead concentrations are on the way. After reaching a peak in 1970, antiknock lead consumption has begun to decline, with a corresponding improvement in ambient air quality. Average national urban air concentration has declined from 1.23 μg/m^3 in 1971 (1.00 μg/m^3 geometric mean) to 0.89 μg/m^3 (0.74 μg/m^3 geometric mean) for the most recent average (1975). The national arithmetic mean is estimated to have declined to 0.58 μg/m^3 in 1978, and is projected to decline to about 0.30 μg/m^3 in 1980 and thereafter, after the target level of 0.5 g/gal lead average in the total gasoline pool is achieved. As developed in Section 8.6, when air lead reaches these levels, inhalation becomes a rather minor contributor to man's body burden of lead. Overall, trends in ambient air lead levels are encouraging, and the limitations now in place are already proving effective.

Lead has a relatively low solubility in water, as low as 30 μg/l in hard water, when analyzed as soluble lead (see APHA Standard Methods and Section 3.1.1 and Figures 3.1 and 3.2). Thus, it is not surprising that most U. S. surface waters contain lead in concentrations below the drinking water standard of 50 μg/l. The extensive 1970 U. S. Geological Survey study found only 3 of 720 samples (0.4 percent) above 50 μg/l. Similar results have been found in surveys of public water systems, and the available data suggest that the median concentration in U. S. drinking water supplies about 10 μg/l. Water quality is not as widely nor as routinely sampled and analyzed as is air quality; there is no network system continuously sampling water analogous to the NASN, which routinely samples air, and hence, annual U. S. averages for water are not available. However, as with air, there is no evidence of an increasing concentration trend in recent years. As compliance with effluent guidelines and other waste disposal regulations improves, lead concentrations in water should decrease.

Although the lead content of city water supplies measured at the reservoirs seldom exceeds 50 μg/l, water drawn from household taps occasionally exceeds this level. This is mainly due to the use of lead water pipes. Low concentrations of dissolved solids (soft water) and an acidic pH exacerbate the problem. Under extremely unfavorable conditions, the lead concentration may exceed 1,000 μg/l, as in Glasgow, Scotland, where lead-lined water storage tanks are an additional problem.

The mean lead concentration in surficial soils in the U. S. is about 16 ppm. Average lead concentrations in soils, except for special cases such as near smelters, heavily travelled highways, or other major lead emission sources, are little above background and are changing only slowly. Lead concentrations in urban dusts, on the other hand, may be quite high, from 1,000 ppm to as much as 11,000 ppm for some house dusts, in extreme cases. Typically, such excessively high lead levels are found in old, deteriorated, sub-standard housing, and most of the lead content of the dusts is attributed to the disintegration of high-lead paint or lead-containing plaster.

It is generally accepted that the major source of lead for the average person is food and beverages. It was, for example, noted in the EPA Air Quality Criteria for Lead document (U. S. Environmental Protection Agency, 1977) that

> "In typical urban settings, food probably constitutes the body's largest direct source of lead because almost every item in the diet contains some measurable amount of the metal."

Neither air nor water compare with food as a source of lead. At an average of 20 m^3 inhaled per day, and the 1975 mean air lead concentration of 0.89 $\mu g/m^3$, total intake from air would be 18 μg; and at 1.5 l of water per day at 20 μg/l, intake from water would be 30 μg. This compares to an estimate of lead intake from food of 254 μg per day for a teenaged male (U. S. Department of Health, Education, and Welfare, 1977). Few people inhale an average air lead concentration in excess of 2 $\mu g\ Pb/m^3$. At worst, this contribution to circulating blood lead concentration is probably somewhere between 2 and 4 μg/dl. Assuming a normal PbB of approximately 15 μg/dl, this leaves about 10-12 μg Pb/dl to be accounted for. In the absence of any other widespread sources of lead intake for the average person, foods and beverages are generally held responsible for the predominant remaining fraction of intake. It is therefore important to consider the extent to which lead in foods and beverages is reducible.

The lead content of most fresh foods is in the range of a few tenths ppm; fresh milk will contain 0.05 ppm or less. However, significant amounts of lead may be introduced in food through processing and packaging, particularly the latter, due to the large role played by canned foods, for which substantial amounts of lead solder are used for sealing seams. It is widely recognized that the soldered seams of cans are potential sources of adventitous lead, and the Acting Director of the Bureau of Foods, FDA, has testified that "about two-thirds of the lead in canned food is from the solder."

Certain foods have been found to double or triple their lead content as a result of the transfer of lead from solder to filler, especially during storage for substantial periods of time. The significance of this transfer is still unknown, however. More information is needed as to: (1) the total dietary contribution of such items and (2) the availability for absorption of the solder contribution.

Hammond (1978) has noted that metallic lead particles such as are found in the side seam region of lead soldered cans are very poorly absorbed compared to lead acetate incorporated into the diet of experimental animals.

And thus, it is quite possible that contamination from solder in the form of microscopic pellets of metallic lead are not nearly as readily absorbed from the gastrointestinal tract as is lead present in foods in the form of simple lead salts bound to natural ligands. There is a need for further studies to establish more clearly the bioavailability of solder-derived lead.

Direct determination of absorption of lead from the digestive tract is not beset with as many technical difficulties as in the case with inhaled lead. Although the number of studies of lead absorption performed in people is extremely limited, the conclusions drawn are fairly consistent. Approximately 8 percent of total, normal dietary lead is absorbed in adults and about 40 percent is absorbed in the case of infants and preschool-age children. Dietary intake of lead in the general adult population is about 150-250 µg/d. Limited information suggests that every 100 µg Pb ingested in the diet daily contributes about 6 µg to blood lead,

In Section 8.6, estimates are developed of the potential benefits of reducing the lead content in canned foods as a means of restricting lead exposure. Various segments of the general population are analyzed as specific examples. Such a reduction in the overall level of lead intake, while not directed at specific "high risk" groups, is projected to have a significant beneficial effect, particularly for those individuals whose current levels of lead ingestion are in the marginally safe range. Since it has been shown in FDA surveys that canned foods contribute approximately 30 percent of total dietary lead intake and at least half, and possibly two-thirds of this 30 percent is attributable to canning itself, realistic savings of approximately 10 to 15 percent of total dietary lead could be achieved if adventitious addition of lead from cans can be prevented or reduced to negligible levels.*

---

* It was learned during the second phase of this investigation that the FDA has a cooperative program underway with the can industry, which is expected to produce drastic reductions in the average lead content of canned foods.

Sample case calculations for the demographic group consisting of 20-34 year old females assuming a two-thirds reduction in lead in canned foods indicated that this would reduce mean blood lead from 15.6 to 15.3 μg/dl. While this would be advantageous for the average individual, perhaps its greater advantage would be in the reduction in the number of persons with blood lead levels at the high end of the spectrum (> 25 μg/dl), in the range where evidence of hemotological changes begin to appear.

A reduction of 1.3 μg/dl does not, at first glance, appear to constitute a significant reduction in average blood lead. However, it represents a nearly 8 percent reduction, and a comparable reduction in lead intake and average body burden is achievable in no other practical way (assuming that air lead has already been taken care of by the new ambient air quality standard for lead (U. S. Environmental Protection Agency, 1978b).

8.2 LEAD IN AIR

As discussed in Section 5.2.2, over one hundred thousand metric tons of lead are being discharged from automobiles to the atmosphere each year, which dwarfs all other discharges. Since this is distributed across the entire United States, there are no areas free from this pollutant, and it is a matter of concern for human health.

Natural sources of lead are trivial in comparison to anthropogenic (man-caused) sources. Natural sources include such items as forest fires and volcanic smokes; most often cited for natural sources of lead are the estimates of Patterson (1965). At that time Patterson estimated that the largest contributor was silicate dust (contributing 5 x 10^{-4} μg/m^3 to the atmospheric total, with volcanic halogen aerosols contributing 3 x 10^{-5} μg/m^3 and forest fire smoke only 6 x 10^{-6} μg/m^3). More recently, Patterson has concluded that the silicate dust figure is too high because it is based on too high an estimate for dust levels. Latest measurement of lead in mid-Pacific air is 2 x 10^{-4} μg/m^3; this lead is in 40-fold excess of crustal silicate values, indicative of anthropogenic enrichment. The same studies suggest a prehistoric natural ambient level of about 4 x 10^{-6} μg/m^3 in mid-ocean air (Patterson, 1978).

For the continental United States, the baseline concentration for atmospheric lead has been suggested by Chow, et al., (1972) to be 0.008 μg/m^3. This value is an annual average, measured at White Mountain, California, in a virtually uninhabited area at an elevation of 3,800 meters, far above the thermal inversion. Patterson (1978) confirms the observation of average lead concentrations of about 0.01 μg/m^3 in the most remote part of the California High Sierras, but notes that this atmospheric lead is enriched about 100-fold above crustal silicate concentrations as a consequence of anthropogenic inputs.

It is suggested by Patterson and his associates on the basis of these data that a prehistoric natural ambient lead concentration may have been about 0.0001 μg/m^3 in continental air near ground level, mainly from silicate soil particles (Patterson, 1978).

8.2.1 Air Lead Concentrations in the U. S.

Only in recent years have ambient atmospheric lead concentrations been measured in the U. S. Occupational atmospheric lead concentrations have, of course, been monitored for many years because of health considerations, and considerable data are available. For the purpose of this discussion, in-plant air lead levels are considered to represent an occupational exposure problem, not causally related to the release of lead to the general environment or to environmental concentrations, and thus, beyond the scope of this study. This matter has been intensively and exhaustively investigated by the Occupational Safety and Health Administration (OSHA) of the Department of Labor, which has promulgated (U. S. Department of Labor, 1978) new lower permissible occupational lead air concentrations in the work place of 50 μg/m^3 (See Section 9.4.1.16).

Illustrative of the relationship of ambient atmospheric lead concentrations to urban population centers and industrial development is the tabulation presented by Harley (1970) of lead concentrations in surface air collected in 1967 by the U. S. Atomic Energy Commission along their 80th Meridan Network (Table 8.1)

Systematic measurements of lead in the atmosphere of a city began in 1941 in Cincinnati, Ohio, but the work was interrupted by World War II and not resumed until the 1950's, at which time a number of U. S. cities were included. The 1941 data from Cincinnati gave an average lead concentration of 5.1 μg/m^3. Cholak (1964) reported that investigations in Cincinnati from 1941 to 1962 showed a continual and gradual downward trend in both mean and median concentrations of lead in air. The mean concentration of 5.1 μg/m^3 in 1941 decreased to 1.43 μg/m^3 in 1962 while the median level fell to 1.27 μg/m^3. After 1954, no lead concentration above 8 μg/m^3 was found. The higher average values and ranges found from 1941 to 1951 may have been partly due to the manner of sampling during this period, and, therefore, were somewhat biased. However, it was concluded by the investigators that the downward trend was real, even though the volume of traffic and consumption of leaded gasoline increased greatly during this period. During the same period, consumption of coal for heating purposes decreased, as did total dustfall (U. S. Department of Health, Education and Welfare, 1965a).

An extensive study to determine the variation in the annual, seasonal, monthly, and diurnal average distributions of lead and total particulate matter in the atmosphere of Cincinnati, Los Angeles, and Philadelphia (3-Cities Study) was carried out by continuous sampling from June 1961 through May 1962 (U. S. Department of Health, Education, and Welfare, 1965a,b). A summary of the

TABLE 8.1 LEAD CONCENTRATIONS IN SURFACE AIR FROM SELECTED SITES ALONG THE 80TH MERIDIAN, 1967[a]

Site Sampled	Latitude	Lead, µg/m^3
Thule, Greenland	70° N	<0.01
Moonsonee, Canada	56° N	0.06
New York, New York	41° N	2.5
Sterling, Virginia	39° N	.74
Miami, Florida	26° N	1.7
Bimini, Bahama Islands	26° N	0.10
San Juan, Puerto Rico	18° N	0.80
Balboa, Panama	9° N	0.23
Guayaquil, Ecuador	2° S	0.35
Lima, Peru	12° S	0.50
Chacahaya, Peru	16° S	0.09
Antofagasta, Chile	24° S	0.06
Santiago, Chile	33° S	0.87
Punta Arenas, Chile	53° S	0.06

[a] Source: Harley (1970). Reprinted, with permission from Environmental Science and Technology. (C) American Chemical Society. 1970

results (Table 8.2) shows that the general urban concentrations ranged from about 1 to 3 µg/m^3 depending on the area sampled. The average concentration of lead for all samples collected during the year was 1.4 µg/m^3 in Cincinnati, 2.5 in Los Angeles, and 1.6 in Philadelphia. The highest average concentration for all samples collected during a single month at any single sampling site was 3.1 µg/m^3 in Cincinnati, 6.4 in Los Angeles, and 4.4 in Philadelphia. The highest individual concentrations were 6.4 µg/m^3 in Cincinnati, 11.4 in Los Angeles, and 7.6 in Philadelphia. A uniform sampling procedure was used in each of the cities, and the samples were analyzed by the same technique.

The results of a subsequent study conducted in 1968-1969 in these same three cities, plus New York, Chicago, Houston, and Washington, D.C. (7-Cities Study) have been described by Tepper and Levin (1975). (See Section 7.5.1). These data indicated higher lead levels at most of the study sites during the more recent of the two periods, consistent with the large increase in tetraethyl lead consumption compared to 1961-62.

The overall situation in the United States with respect to atmospheric lead was well summarized by the report on airborne lead by the National Academy of Sciences National Research Council Committee on Biological Effects (1972), and their findings are still valid. As pointed out in that report, the concentration of lead in ambient air is closely correlated with the density of vehicular traffic, being highest in large cities, lower in suburban areas, and lowest in rural areas. These gradients are also subject to diurnal and seasonal cycles, primarily as a result of meteorological differences.

Although the basic situation remains much the same as when the NAS-NRC report was prepared, results of other investigations have appeared which further elaborate the findings reported therein, and sampling and analysis have improved considerably in the intervening years, so that an extension and update is appropriate.

It has only been in recent years that much attention was paid, on a national basis, to trace metal concentrations in the atmosphere. Prior to this, analytical efforts tended to be localized and related to specific actual or suspected problems. Beginning in 1953, an air quality sampling program was begun under the auspices of the U. S. Health Service, albeit on a much more limited scale than today's efforts. This sampling effort was ultimately taken over by the U. S. Environmental Protection Agency after its' formation, and is now designated the National Air Surveillance Network (NASN), which routinely monitors air pollutant concentration levels in urban and nonurban America. Principal concern has been with the tracking of changes in fossil fuel combustion products (particulates and SO_2) and in the automobile-related "criteria" pollutants, but data are now collected routinely on 11 metals, one of which is lead. Air particulate samples have been collected since 1970 at approximately 300 urban and 35 nonurban sites (some of the latter have taken on the characteristics of urban sites with time and expanding urban development). National cumulative arithmetic and geometric means for the 1970-1975 period have been reported by EPA (Akland, 1976; U. S. Environmental Protection Agency, 1978a),

TABLE 8.2 CONCENTRATION OF LEAD IN THE ATMOSPHERE OF THREE CITIES, 1961-1962

	Atmospheric Lead Concentration, μg/m^3		
	Cincinnati	Los Angeles	Philadelphia
Annual average values			
Downtown	2	3	3
Outlying area	1	2	1
All stations	1.4	2.5	1.6
Seasonal distributions (all stations):			
Summer	1.3	1.9	1.4
Fall	1.7	2.8	1.9
Winter	1.3	3.1	1.9
Spring	1.3	2.1	1.4
Diurnal distributions (annual--all stations stated as a fraction of annual mean):			
2300-0300	1.3	1.0	0.9
0300-0700	1.1	1.1	0.8
0700-1100	1.4	1.2	1.4
1100-1500	0.8	0.7	0.8
1500-1900	0.9	0.8	1.1
1900-2300	1.0	1.1	1.1

[a] Adapted from U.S. Department of Health, Education and Welfare (1965a).

as shown in Table 8.3. The improving quality of urban ambient air with respect to lead concentrations, since the peak year of 1971, is evident in these data. For any given cumulative frequency, the lead level each year is lower, and there has been a steady decline in the annual mean concentrations. In 1974 and 1975, the most recent years for which data are available, the annual geometric means were 0.74-0.75 μg/m^3 (0.89 μg/m^3 arithmetic mean); and almost 90 percent of the 1,300 urban quarterly averages were below the recently-promulgated 1.5 μg/m^3 ambient air quality standard for lead (U. S. Environmental Protection Agency, 1978b).

The results from the 35 non-urban sampling sites indicate an arithmetic mean for 1975 of 0.085 μg/m^3 (0.054 μg/m^3 geometric mean), about a factor of ten lower than the comparable urban average. The nonurban means appear to have varied somewhat randomly during the 1970-1975 period; there has not been a steady improvement as shown in the urban average. These nonurban means may represent the minimum, ultimately attainable, for urban air lead concentration averages.

In the discussion of requirements for state implementation plans to comply with the 1.5 μm^3 ambient air quality standard (a standard not to be exceeded on a quarterly basis), 31 urban areas were cited by EPA as having had quarterly air lead concentrations equalling or exceeding 1.5 μg/m^3 in 1975 (U. S. Environmental Protection Agency, 1978). These areas are listed in Table 8.4, along with 1975 maximum quarterly and annual (where available) averages published in the 1975 air quality data for metals (Rhodes and Fair, 1978). There appear to be some discrepancies; a number of urban areas, e.g. Birmingham, Alabama, Philadelphia, Pennsylvania, and Washington, D.C., are not recorded as having exceeded 1.5 μg/m^3 in any quarter in 1975.

The "rollback" needed to bring many of these urban areas into compliance is moderate, and is probably attainable with the planned lead phasedown. Reduction in lead consumption from 1975 reached 17 percent by 1978 (Table 4.16), should reach 26 percent by 1979, and 60 percent by 1980 (Table 4.18), by which time, almost all of the listed areas should be in compliance.

8.2.2 Localized Source-Oriented Studies

A considerable number of localized source-oriented studies of ambient air lead concentrations have been conducted since the 1950's. Many of these have been aimed at various facets of antiknock emissions and their influence on close-in "microclimates". Others have attempted to develop lead concentration profiles in the vicinity of point sources such as smelters.

8.2.2.1 Antiknock-Related Studies--

Illustrative of the "microclimate" type of study was the one by Daines, et al., (1970), who investigated the lead content of the air as a function of traffic volume, proximity to the highway, wind direction, and acceleration versus constant speed. One of their principal findings was that air lead levels near busy (New Jersey) highways are relatively high, 2 to 7 μg/m^3, at 10 meters distance, but drop off rapidly within a short distance (50 meters), beyond which the rate of decrease is gradual. As shown by Figure 8.1, at 180 meters (500 feet), ambient air concentration had decreased to the 1-2 μg/m^3 range.

TABLE 8.3 NATIONAL AVERAGE AMBIENT ATMOSPHERIC LEAD CONCENTRATIONS: QUARTERLY COMPOSITES. (μg/m^3)[a]

Year	Number Stations	Minimum Reported Value	Cumulative Frequency Distributions, Percent							Max.	Arithmetic		Geometric	
			10	30	50	70	90	95	99		Mean	Std. Dev.	Mean	Std. Dev.
							Urban							
1970	797	LD[b]	.47	.75	1.05	1.37	2.01	2.59	4.14	5.83	1.19	.80	.99	1.84
1971	717	LD	.42	.71	1.01	1.42	2.21	2.86	4.38	6.31	1.23	.87	1.00	1.89
1972	708	LD	.46	.72	.97	1.25	1.93	2.57	3.69	6.88	1.13	.78	.93	1.87
1973	559	LD	.35	.58	.77	1.05	1.62	2.08	3.03	5.83	.92	.64	.76	1.87
1974	594	.08	.36	.57	.75	1.00	1.61	1.97	3.16	4.09	.89	.57	.75	1.80
1975	695	LD	.37	.58	.78	.96	1.54	2.02	3.15	4.94	.89	.59	.74	1.82
							Non Urban							
1970	124	LD	LD	LD	LD	LD	.267	.383	.628	1.471	.088	.190	.040	3.72
1971	85	LD	LD	LD	LD	LD	.127	.204	.783	1.134	.047	.155	.008	4.80
1972	137	LD	LD	LD	.107	.166	.294	.392	.950	1.048	.139	.169	.090	2.59
1973	100	LD	LD	LD	LD	.132	.233	.392	.698	.939	.110	.149	.068	2.77
1974	79	LD	LD	.053	.087	.141	.221	.317	.496	.534	.111	.111	.083	2.30
1975	98	LD	LD	LD	LD	.144	.255	.311	.431	.649	.085	.126	.054	2.95

---

[a] Source: Rhodes and Fair, (1978).

[b] Limit of Detection

TABLE 8.4 LIST OF 31 URBAN AREAS WITH QUARTERLY AIR LEAD CONCENTRATIONS EQUALLING OR EXCEEDING 1.5 $\mu g/m^3$ in 1975[a]

Urban Area	1975 Data [b]		Rollback Needed, Percent
	Average	Max Q	
Birmingham, Ala	0.68	0.89	--
Phoenix, Ariz	2.07	3.16	53
Fresno, Cal	0.95	1.59	6
L.A.-Long Beach, Cal	2.45[c]	3.45[c]	57
Sacramento	0.90	1.61	7
San Bernadino	1.37	1.71	9
San Diego	1.36	2.64	43
San Francisco-Oakland	1.45[d]	2.31[d]	35
Denver, Colo	--	1.75	15
NY, NY-N.E. N.J.	0.93	1.09	--
Waterbury, Conn.	1.52	1.74	18
Springfield, Chicago	--	1.50	--
Wilmington, Del-NJ	1.13	1.57	5
Philadelphia, Pa - NJ	1.07	1.15	--
Wash, D.C. - MD, Va	--	1.08	--
Chicago - N.W. Indiana	2.69	4.61	68
Minneapolis - St Paul	2.16	3.24	54
St. Louis -Ill	--	1.73	13
Las Vegas, Nev	1.81	2.91	49
Reno, Nev	1.18	2.15	30
Oklahoma City (1974)	1.92	3.75	61
Scranton, Pa	1.89	2.28	35
San Juan, Pr	1.43	1.93	23
Columbia, SC	1.00	1.85	19
Greensville, SC	1.08	1.52	1
Knoxville, Tenn	0.77	1.20	--
Memphis, Tenn	1.82	1.99	25
Dallas, Tex	2.77	3.03	51
El Paso, Tex	1.57	2.34	36
Houston, Tex	1.91	2.25	33

[a] Source: 43 FR 46269, October 5, 1978.

[b] Data from Rhodes and Fair (1978).

[c] Sampling station Torrance, Cal.

[d] Sampling station San Jose, Cal.

At 3 meters (10 feet) from a highway carrying 58,000 cars/day, average annual lead concentration was 10 μg/m^3. This was reduced about 50 percent at a distance of 10 meters (30 feet).

Another conclusion of this study was that over 65 percent of the lead in the air from 10 to 530 meters (30 to 1750 feet), from a well-traveled highway, consisted of particles under 2 μm in diameter and over 85 percent consisted of particles under 4 μm. These fall, unfortunately, in the 0.1 to 10 μm particle size range shown by Natusch and Wallace (1974) to have maximum pulmonary deposition.

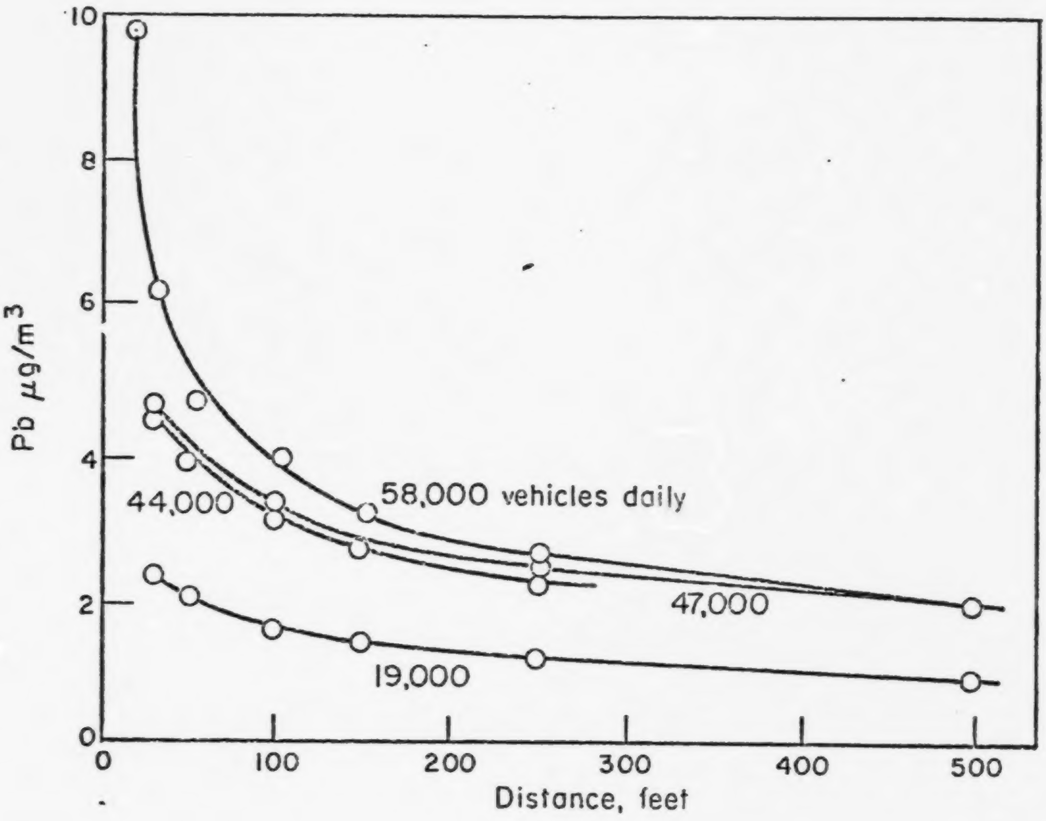


Figure 8.1 Air lead values as a function of traffic volume and distance from the highway.

Source: Daines, et al. (1970). Reprinted with permission from Environmental Science and Technology. (c) American Chemical Society. 1970.

In a later similar study, Daines, et al., (1972) compared the lead content of air inside and outside homes located close (3.7 meters) to a busy New Jersey street with similar homes at greater distances, 38 and 122 meters (125 and 400 feet). Continuous air samples were taken on front porches and in front rooms inside the homes for 2 years; blood samples were also taken on two occasions. Results of this study, summarized in Table 8.5 show that air samples taken inside homes were consistently less (an average of about 45 percent) than those taken out of doors. There were no significant differences between the indoor air samples at the three distances except during the open window (summer) season. The outdoor samples at homes 3.7 meters distant from the highway were consistently greater than at those at 38 to 122 meters. Use of a room air conditioner significantly reduced average indoor lead concentration at the close-in homes, to a level comparable to the indoor concentrations in the more distant homes. (Biological implications of this study are discussed in Section 7.5.2.1).

Several recent similar "microclimate" studies have been conducted to assess the effects of lead emissions from gasoline antiknock on crops and soils; these are discussed in the sections on soils (8.4) and crops (6.3.2).

Concern about levels of alkyllead compounds in the air have prompted several localized studies of situations where gasoline is transferred, or burned in confined spaces, as well as of general urban ambient levels. Purdue, et al., (1973) described the organic lead concentration in an underground parking garage and in the general ambient air of six major cities in the U. S. In the parking garage, the total air lead level was 11.7 μg/m^3, of which 16.7 percent was organic lead. In the six major cities, the organic lead concentration was about 10 percent of the total lead (about 0.2 μg/m^3). Differences found in the concentration of organic lead relative to particulate lead may be related to differences in proximity to the emitting sources such as gasoline stations and gasoline storage centers. Also, tetraethyllead and tetramethyllead undergo rapid photochemical decomposition in the atmosphere, which can affect alkyllead concentrations. Colwill and Hickman (1973) and Harrison, et al., (1974) found alkyllead concentrations of 0.2 to 1.5 μg/m^3 in the vicinity of gasoline pumps, and the concentration of tetraalkyllead emitted from the exhaust pipe of cars varied from 50 to 1000 μg/m^3 when the engine was idling (Laveskog, 1971).

8.2.2.2 Smelter-Related Studies--

Other localized "microscale" investigations have been conducted in the vicinity of known or suspected "hot spots", quite often smelters. East Helena, Montana is the site of a lead smelter which has been operating since 1888. During the summer and fall of 1969, a source-oriented study of the Helena Valley, Montana area was undertaken using dustfall bucket and high-volume sampling techniques (U. S. Environmental Protection Agency, 1972). During this period, the city of Helena was exposed to an average daily concentration of 0.1 μg/m^3 with maximum concentrations up to 0.7 μg/m^3. The residents of the East Helena area were exposed to an average daily concentration of 0.4 to 4.0 μg/m^3, depending upon proximity to the source, with maximum daily exposures up to 15 μg/m^3. Within a 1.7 km (1 mile) radius of the smelter, the fallout of lead on

TABLE 8.5 EFFECT OF PROXIMITY TO HIGHWAY IN AN URBAN ENVIRONMENT ON AIR AND BLOOD LEAD CONCENTRATIONS[a]

Time of Year	Location of Sampler	Pb Concentration, $\mu g/m^3$ Area A 3.7 m	Area B 38.1 m	Area C 122 m
Nov - April	Front porch	4.56	2.63	2.37
	Front room	1.85	1.55	1.67
May - Oct	Front porch	4.68	2.05	2.07
	Front room	2.37	1.40	1.43
Entire year	Front porch	4.60	2.41	2.24
	Front room	2.30	1.50	1.57
		Pb Concentration, µg/100 g blood[b]		
April sample		22.3	16.9	17.9
November sample		23.9	17.9	17.4
Average		23.1	17.4	17.6

[a] Source: Adapted from Daines, et al. (1972). Reprinted with permission from Environmental Science and Technology (c) American Chemical Society, 1970.

[b] Blood from women living in homes.

the ground ranged from 30 to 140 mg/m^2; an area of from 0.05 to 0.18 m^2 would contain the body burden limit for lead.

In a more recent study, an intensive investigation was conducted around another old smelter, the one at El Paso, Texas which has been in operation since 1887. This copper-lead-zinc smelting complex has been the subject of studies of lead in blood, dust, soil, and air, with the conclusion that the smelter was producing elevated levels of lead in all of these media for a distance of 0.6 kilometers and to lesser degrees in various media for up to 6.6 kilometers (Landrigan, et al., 1975). Epidemiological aspects of this study are discussed in Section 7.5.2.2.1.

The El Paso City-County Health Department estimated in 1971 that the emissions from 1969 through 1971 had comprised 1010 metric tons of lead, 508 of zinc, 11 of cadium, and 1 of arsenic. The pollution problem was accentuated by the topography and meteorology of the area, which is surrounded by high mountains and is extremely arid (10 to 25 cm of rainfall per year), so that fine dust is frequently present. Thermal inversions occur on 70 percent of the mornings. Air samples were collected upwind and downwind of the smelter at the property boundary, 60 to 120 meters from the base of the main stack, and at 39 other locations in El Paso. The annual mean lead concentration at the property boundary downwind of the smelter in 1971 was 92 $\mu g/m^3$, with the range from 15 to 269 $\mu g/m^3$. Levels fell rapidly with distance, and reached background values at 4 to 5 km (Figure 8.2). From June, 1972 through July, 1973, the mean lead concentration was again highest downwind of the smelter (43 $\mu g/m^3$), a considerable reduction over the 1971 values, but still excessively high (Landrigan, et al., 1975).

Dustfall was also high, especially near the smelter (204 $mg/m^2/mo$), decreasing with distance. As a result, children in the first 1.6 km with blood levels of 40 μg/dl or over were found to be exposed to dust with concentrations of 6450 ppm lead; children with lower blood levels were exposed to dusts of 2070 ppm lead. High concentrations (around 1 percent by weight) of lead in the soil around the smelter were attributed partly to the fact that the smelter had been operating for 85 years.

Other aspects of possible exposure were also checked during this investigation, including food, water, house paint, pottery soil, and dust. The data appear to be conclusive in suggesting that particulate lead in dust and air accounted for most of the lead absorption in El Paso children, and that the smelter was the principal source of the lead. In 1977, a multimillion-dollar program of control measure was undertaken, including improved control of fugitive emissions and collection of surface soil in the neighborhood of the plant.

The "New Lead Belt" in southeastern Missouri has also been investigated to determine the effect of the lead mining and smelting operations on air quality and distribution of pollutants. Since the commissioning of the first mine in 1969, this industrial district has become one of the world's largest lead-producing areas, mining more than 392,277 metric tons of lead, or 75 percent of the entire U. S. lead production during 1970 (U. S. Bureau of Mines, 1976). Annual averages for suspended lead collected in Glover, Missouri, by high-volume samplers were 3.4, 5.3, and 5.5 $\mu g/m^3$ in 1970, 1971, and 1972 respectively (U. S. Environmental Protection Agency, 1977b).

Other similar studies have been conducted in Omaha, Nebraska, and in Southern Solano County, California; the results of these are summarized in a recent EPA document on air quality criteria for lead (U. S. Environmental Protection Agency, 1977b).

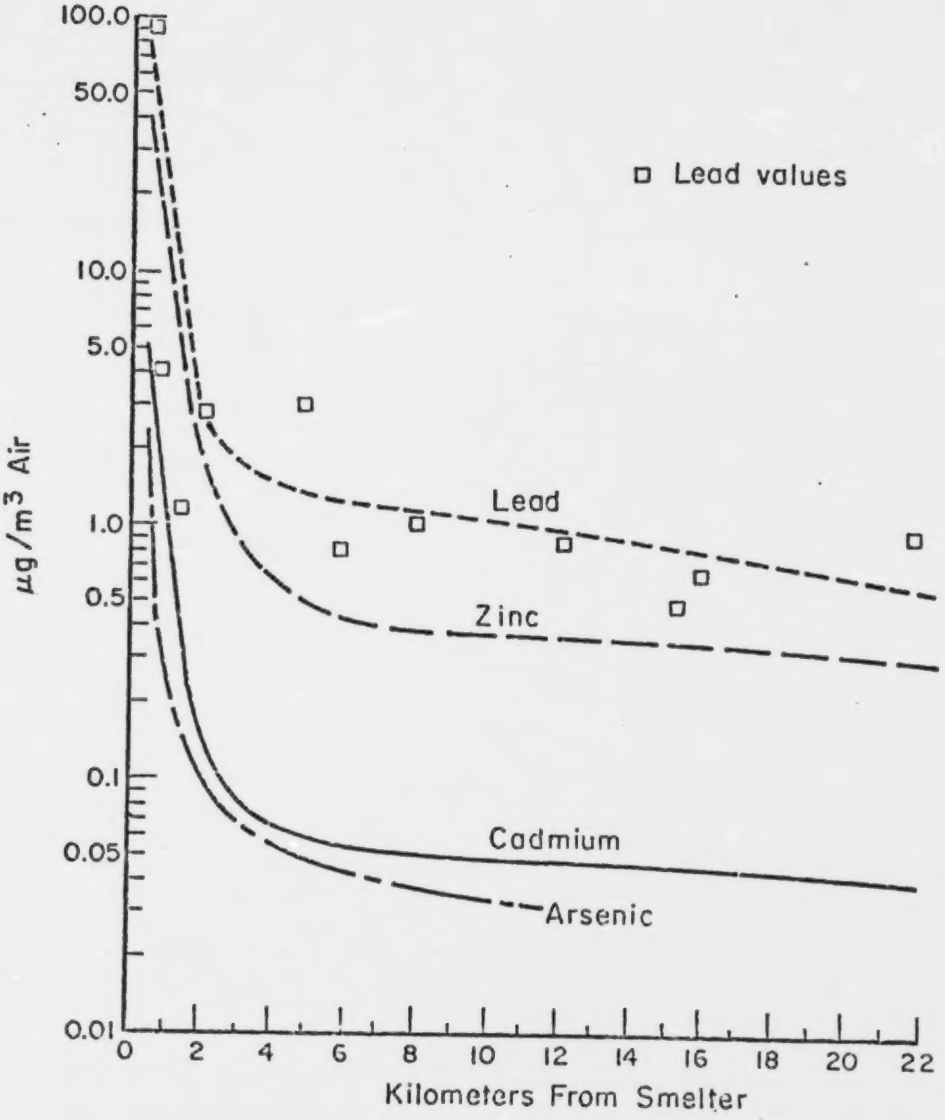


Figure 8.2 Concentrations of metals in air as a function of distance from the El Paso smelter.

Source: Landrigan, et al.. (1975). Reprinted, by permission, from the New England Journal of Medicine (1975).

8.2.3 Trends in Atmospheric Lead

From their introduction till consumption reached a peak in 1970 (Table 4.16), quantities of alkyllead antiknock additives increased annually. Now that this dominant source of lead in the atmosphere has finally begun to decrease, it seems inevitable that ambient lead air concentrations will also similarly decrease.

The mass of detailed data in the NASN files for 1965 through 1974 was correlated and analyzed by Faoro and McMullen (1977). For this analysis, criteria for selection of sample stations were length and continuity of record, in order to maintain the comparison on a consistent basis. This reduced the data base to 92 NASN urban sampling sites, a small number on which to base either regional or national averages; also, because of the selection of the sample population for this analysis, the averages are not totally comparable with the national averages and means for all stations (Table 8.3). Nevertheless, they provide useful measures of relative rather than absolute concentrations across time, and illustrate national trends in air lead concentrations.

The composite 50th percentile of lead concentrations at urban sites increased steadily after 1965, peaking at a little over 1.1 $\mu g/m^3$ in 1971 and then declined, reaching 0.85 in 1974, about the level it had started from in 1965. This was a statistically significant decrease of about 24 percent, with most of it occurring between 1972 and 1973. This general trend was described as being consistent at most sites studied. It was found that ambient air lead concentrations were about 30 percent higher in the Far West (where California sampling sites predominate) and in the Northeast, than in the other three geographic regions of the country. This is not surprising, and appears to correlate with the respective vehicle densities.

In another paper, McMullen and Faoro (1976) described the results of an analysis of data covering 11 metals obtained on airborne particulate matter samples from the NASN for the period 1965-1974. The analysis considered both the concentrations per unit volume of air and the relative abundance compared to crustal abundance. The frequency distribution curve (Figure 8.3) shows that while the average of urban mean air lead concentrations was approximately 0.75 $\mu g/m^3$, about 30 percent of the analysis exceeded an average of 2 $\mu g/m^3$, with some of these as high as 6 $\mu g/m^3$. The mean of the nonurban sites was 0.04-0.05 $\mu g/m^3$, with lead at the limit of detection for 30 percent of the stations; and the maximum was less than 0.5 $\mu g/m^3$.

McMullen and Faoro also made the interesting comparison of abundance of lead in particulate samples versus the average and range of lead contents in soils, as reported by Shacklette, et al., (1971) in their very extensive survey of U. S. soils. Urban particulate samples had a mean lead content of approximately 10,000 ppm (1 percent) compared to the average soil lead content of about 16 ppm (Figure 8.4).

Whereas the nonurban particulate samples had abundances of other metals

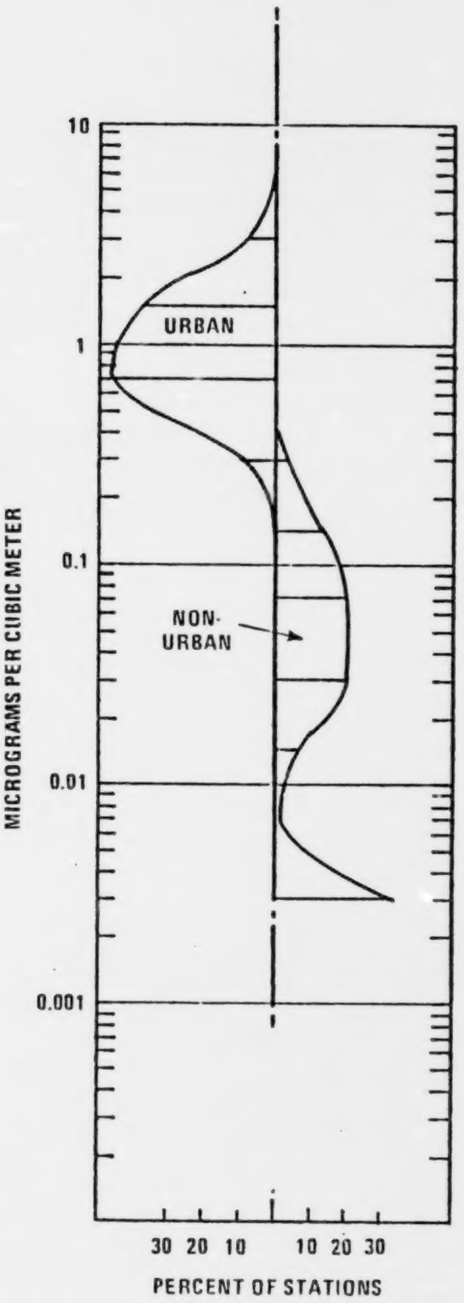


Figure 8.3 Concentration of particulate lead in air.

Source: McMullen and Faoro (1977)

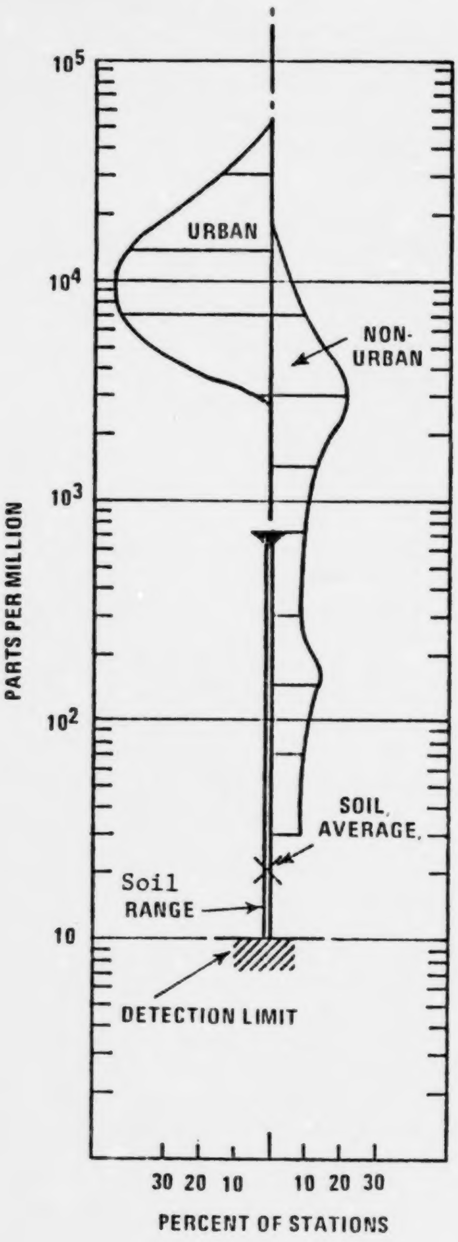


Figure 8.4 Relative abundance of lead in airborne particulates and in soil samples.

Source: McMullen and Faoro (1977)

quite comparable to their abundances in soils, lead was an exception; the lead content of nonurban samples was in some instances, up to the 1 percent level. This was attributed to the fact that the data were significantly influenced by the results from the station located at the Grand Canyon Visitors Center. While this is a nonurban site, it is one with a high density of automobile traffic and one which illustrates the influence of antiknock emissions on ambient air lead concentrations.

The 0.5 g/gal maximum lead content for the entire gasoline pool, mandated by EPA to be achieved by October 1, 1979 will reduce annual lead consumption for alkyllead antiknock additives to an estimated 63,000 to 67,000 metric ton range in the 1980-1985 period (See Table 4.18).

The estimated effect of this decrease on average ambient air lead concentrations is graphed in Figure 8.5. Historical air lead values are geometric means from Table 9.3; historical lead consumption is from Table 4.16 and projected future lead consumption based on the present phasedown schedule is from Table 4.18.

On the basis of the data for the six-year period between 1970 and 1975 (and adopting the conservative assumption that all of the lead measured in the air resulted from the combustion of leaded gasolines) each 1000 metric ton charge in antiknock lead consumption produces a change of 0.0043 $\mu g/m^3$ in mean air lead concentration. This relationship may be used to project national average ambient air lead concentrations beyond 1975. For 1976-1978, lead usage is taken from Table 4.16. Future lead usage (Table 4.18) is estimated from the gasoline consumptions projected by the ESCON model (E.I. du Pont de Nemours and Co., Inc., 1978), summarized in Table 4.17.

For the base case, lead levels in the gasoline pool are assumed to continue at the 1978 average of 1.20 g/gal until October 1, 1979, and at the EPA-mandated level of 0.59 g/gal thereafter. Projected annual average ambient air lead concentrations are estimated to have decreased to 0.58 $\mu g/m^3$ in 1978, to further decrease to 0.52 $\mu g/m^3$ in 1979, and to 0.29-0.30 $\mu g/m^3$ in 1980 and thereafter.

If, on the other hand, lead contents of leaded gasolines continue to follow present industry practice, and contain no more than about 2.00 g/gal lead in regular and 2.50 g/gal in premium (See Table 4.15), the pool average will fall considerably below 0.59 g/gal as cars using leaded gasoline are replaced by cars requiring unleaded gasoline. By 1990, the pool average could be as low as 0.30 g/gal and total lead consumption no more than 29,500 metric tons/yr. (Table 4.18). Under these conditions, an average urban air lead concentration of 0.13 $\mu g/m^3$ is projected.

While national annual average ambient air lead concentrations can be correlated with total annual lead antiknock consumption, there are no similar direct relationships which can be employed to estimate or predict air lead concentrations for smaller areas. Site-specific air quality calculations can be performed for very small areas, provided that a suitable validated mathemat-

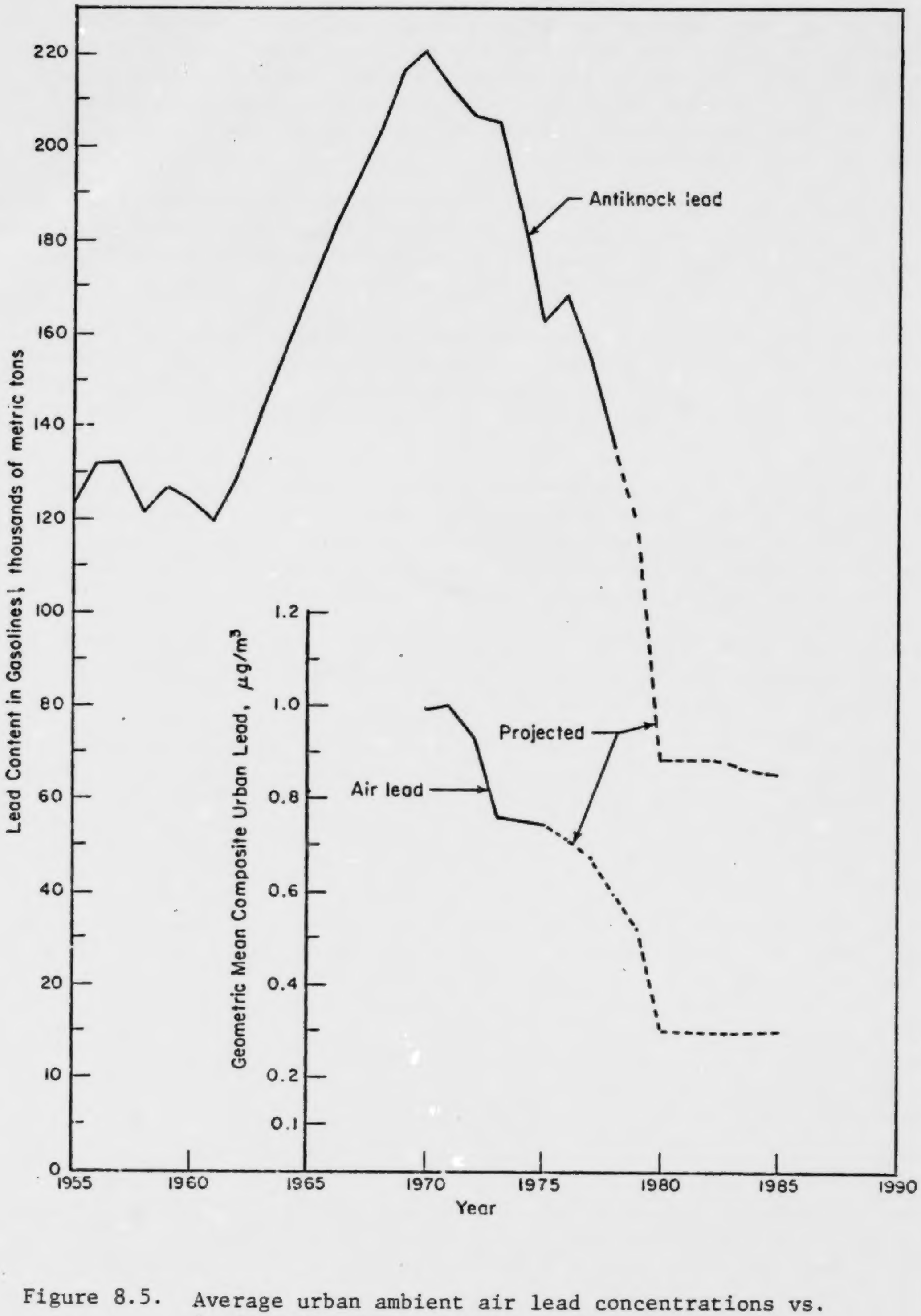


Figure 8.5. Average urban ambient air lead concentrations vs. lead antiknock consumption, with phasedown.

Source: Battelle-Columbus estimates.

ical model is available.

Numerous variables besides quantity of emitted lead can significantly influence air lead concentrations. It is, for example, well-known that air lead concentrations are inversely proportional to distances from highways, and that concentrations at 100 meters may be one-half to one-third or less those at 10 meters, e.g. see Figure 8.1, so that location of the measurement point is very important. Topography, and its effects on local air movement can similarly be quite important. Seasonal differences, e.g., as in California, can have major effects upon the air lead concentrations resulting from unit lead emission. Illustrative is a comparison of the 1975 quarterly air lead averages in Northern California with quarterly gasoline consumption. Although 1975 gasoline consumption data are not available, the 1976 data reported in Highway Statistics, 1976 (U. S. Department of Transportation, 1977) may be considered representative:

	1976 Gasoline Consumption, Billions of Gallons	1975 Average Air Lead, μg/m^3[a]
Q1	2.763	1.15
Q2	2.967	0.56
Q3	2.995	0.71
Q4	2.960	1.62

[a] Average of Sacramento, San Francisco, Oakland, Berkeley, San Jose, and Fresno

The spring and summer quarters had slightly larger gasoline consumptions than the fall and winter quarters, but air lead concentrations were less than one-half as great.

Thus, it is apparent that a straight rollback may not be appropriate for all areas, and seasonal modifications may be appropriate for some areas where climatic or topographic considerations dictate.

As emissions from leaded gasolines decrease, so also will secondary impacts derived from lead alkyl antiknock additives, such as lead in dustfall on food crops, in nonpoint source runoff from the urban areas, and from the combustion of used lubricating oil. There will be temporary aberrations, certainly, most likely in those urban centers of highest density but, overall, the prognosis appears extremely favorable.

The recently promulgated national ambient air quality standard for lead of 1.5 μg/m^3 (U. S. Environmental Protection Agency, 1978b), will provide the mechanism for controlling the few remaining localized point sources, e.g., existing battery plants and primary and secondary lead smelters, which have not been regulated by New Source Performance Standards.

Overall, the effectiveness of the steps already taken suggest that, in general, few additional limitations on atmospheric lead emissions are necessary at this time.

8.3 LEAD IN WATER

The lead profiles in the major oceans of the world were determined by Chow (1968), who used mass spectrometer techniques to analyze samples from various depths as well as locations. The results indicated that there are no essential differences in lead concentration in deep waters below the 1000 meter level, but in the surface layers, lead is present in greater amounts in the Pacific and Mediterranean waters than in the central Atlantic, away from land influences. Chow also cited data by other investigators (Table 8.6) which show the wide range of values found in different places, from 0.02 micrograms per kilogram (ppb) in ocean depths to 8 micrograms per kilogram in surface waters off Europe.

TABLE 8.6 LEAD CONTENT OF VARIOUS MARINE WATERS[a]

Source of Seawater	Concentration, ppb
Florida Key	3-5[b]
North Sea--Surface	2
Brittany-Surface	3-8
Gullmarfjord-Surface	5-8
Japan Coast-Surface	4
English Channel-Surface	5
Washington Coast-to 100 m	0.1
English Channel	0.6 -1.5
Atlantic-to 5,300 m	0.02-0.10
Pacific-to 4,000 m	0.02-0.35
Bermuda-to 3,000 m	0.03-0.07

[a] Source: Chow. Reprinted with permission from Journal of Water Pollution Control Federation. (c) Water Pollution Control Federation, 1968.

[b] Limit of sensitivity of method.

According to Patterson and associates, many of the previous analyses of low concentrations of lead in ocean water have been seriously in error through contamination. Schaule and Patterson (1978) have determined the lead profile in the mid-Pacific, which, while in general agreement with the decreasing concentration with depth described by Chow, found lead concentrations to be considerably lower. The lead profile essentially parallels those of unsupported lead-210 and tritium, as shown in Figure 8.6. The concentrations do not change significantly to depths of 250 to 400 m, then decrease sharply within small depth intervals to very small values, after which concentration changes become more gradual. Below a couple thousand meters dissolved lead concentrations seldom exceed 0.002 µg/kg, only a tenth of the value reported by Chow. Mass balance considerations indicate much of the lead introduced to the oceans enters from the atmosphere, primarily from the combustion of leaded gasolines.

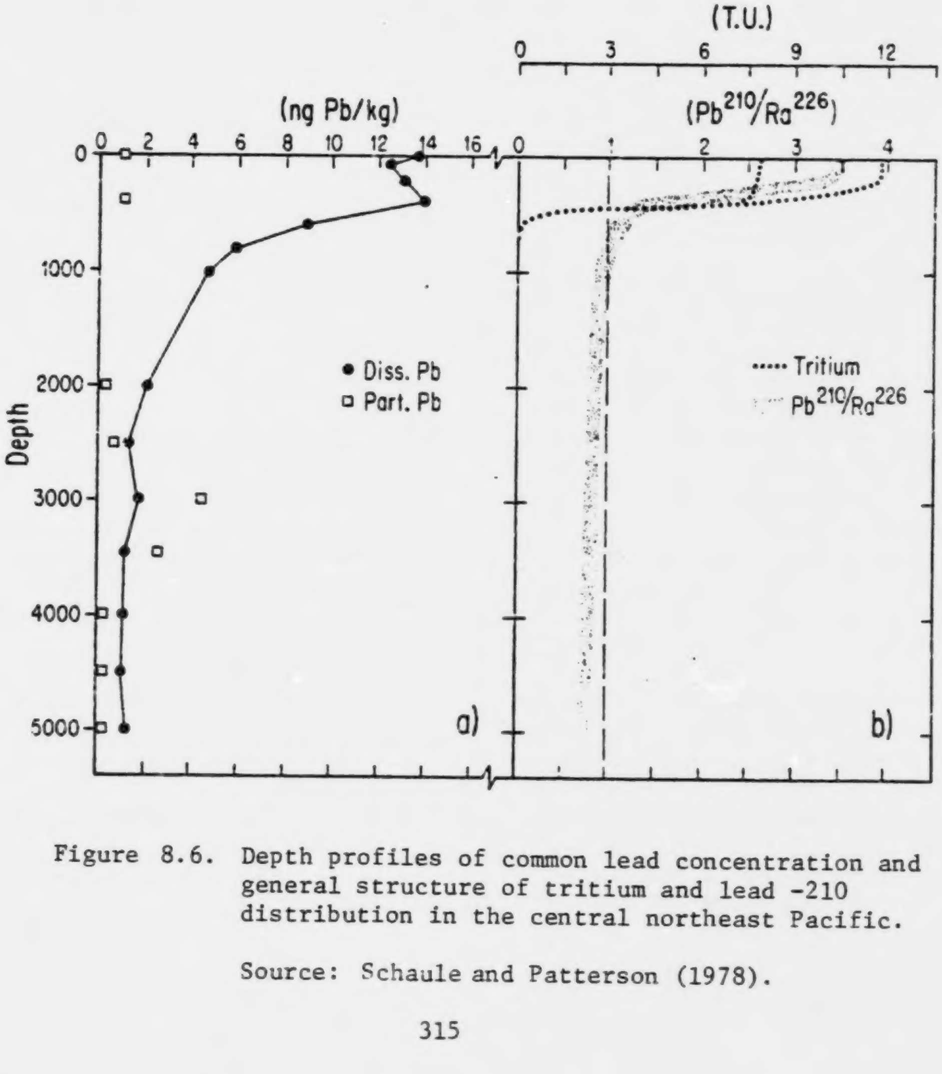


Figure 8.6. Depth profiles of common lead concentration and general structure of tritium and lead -210 distribution in the central northeast Pacific.

Source: Schaule and Patterson (1978).

Since both tritium, as debris from nuclear bomb tests, and unsupported lead^{210}, derived from continental radon-222 emanations, enter by basically the same route, the similarity in profiles is not surprising (Schaule and Patterson, 1978).

8.3.1 Water Lead Concentrations in the U. S.

The concentrations of lead in U. S. waters are generally low, almost uniformly below the 50 µg/l (ppb) limit specified by the Interim Drinking Water Standard (U. S. Environmental Protection Agency, 1975g). The generally low values found for dissolved lead reflect the relative insolubility of the forms (hydroxide, carbonate and hydroxy carbonate) likely to result from ambient conditions. As noted in Section 3.1, equilibrium calculations indicate that the total solubility of lead in hard water is about 30 ppb, and 500 ppb in soft water (Davies and Everhart, 1973).

A number of surveys of heavy metals in raw and finished waters of the U. S. have been conducted. One of the broadest geographically was the one investigating levels of heavy metals in lakes and rivers of the U. S. conducted by the U. S. Geological Survey during autumn dry-weather flows in 1970 (Durum, et al., 1971). More than 720 samples were obtained, in three categories:

- Surface water sources for cities exceeding 100,000 population (or largest city in state)
- Water courses downstream of major municipal and/or industrial complexes in each state
- USGA hydrologic bench-mark stations (located near headwaters of undeveloped drainage basins).

Samples were filtered before analysis (0.45 micron openings) to provide information on sediment-free water like that normally supplied to the user; thus, essentially only dissolved lead was measured. The results of this large reconnaissance, as summarized by Durum (1974), were that detectable concentrations of lead (> 1 µg/ℓ) were found in 63 percent of the river water samples. Only three values exceeded 50 ppb; however, there was a distinct regional pattern in the data as shown in Table 8.7. A larger proportion of the waters from the northeastern and southeastern states contained lead above the detection limit, and the largest share of the higher concentrations, i.e., > 10 ppb. Median lead concentration for 727 samples was 2 ppb.

In some mineralized areas in the western state where water is scarce, higher lead concentrations have been observed, as indicated by Figure 8.7, from the 1976 Annual Report of the Council on Environmental Quality (1976).

The U. S. Geological Survey maintains a computerized data bank for six toxic substances, including lead, in water. Concentrations are determined for the metals after filtration through a 0.45 micron filter (dissolved phase) and in water containing suspended sediment (total phase). Dissolved values for

TABLE 8.7 REGIONAL SUMMARY OF LEAD IN SURFACE WATERS OF THE U. S.[a]

Region	Concentration Range, ppb Maximum	Minimum	Median	< 1 ppb, percent
New England and Northeast	890	<1	6	8
Southeastern	44	<1	4	27
Central	84	<1	1	51
Southwestern	34	<1	1	61
Northwestern	23	<1	1	62

[a] Source: Durum and Hem. 1972. Reprinted, with permission, from Annals of New York Academy of Science. (c) New York Academy of Science (1972).

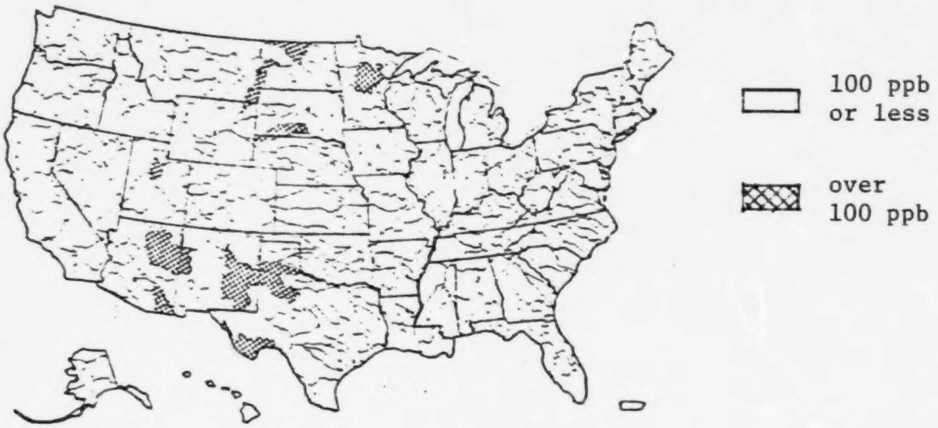


Figure 8.7. Total levels of lead in U. S. streams: 1975
Source: Council on Environmental Quality (1976).

lead are compared to the 50 ppb primary drinking water standard; "total values are compared to the 30 ppb recommended for freshwater aquatic life and wildlife. The data for lead concentrations in surface waters in the computer storage up to mid-January, 1976, are presented in Table 8.8. It is apparent from these data that while the northeastern and southeastern states might have more values above 10 ppb, as noted by Durum, those with more than a nominal number in excess of 50 ppb were states with significant lead mineralization, e.g., Colorado, Missouri, and Washington. The reason for Florida's inclusion in this list is unknown. Approximately 86 percent of the samples had detectable lead concentrations (> 1 ppb), and 3.3 percent exceeded 50 ppb.

Analysis of 3,266 samples of ground water showed 65 percent (2,145) with a detectable concentration of lead, and 2.9 percent (94) which exceeded 50 ppb. Colorado was again in the group with a significant number of high values; Pennsylvania and New Jersey also were in this group.

The potential sources for elevated lead concentrations in surface waters are many and varied. Rain water can be a direct source of lead to surface waters and sediments. As shown by Lazrus, et al., (1970), rain water can average 34 ppb, and reach values as high as 300 ppb. Rain water can also be an indirect source by washing lead-containing dustfall from roofs and streets into surface water courses; some as dissolved lead or, more likely, as particulate matter. Since most of the lead discharged from vehicle exhausts appears to fall out in the near vicinity of its discharge, urban surfaces are the initial receptors of much of the lead from gasoline. Thus, it is not surprising that

TABLE 8.8. NUMBER OF SAMPLES OF SURFACE WATERS EXCEEDING SPECIFIED CONCENTRATIONS OF LEAD[a,b]

	Dissolved				Total			
State	N	N>0	N>50 ppb	Max. ppb	N	N>0	N>30 ppb	Max. ppb
Ala.	97	93	2	100	33	33	18	100
Alaska	113	95	4	130	72	67	53	6,500
Ariz.	21	19	1	100	12	12	12	300
Ark.	41	41	0	50	148	146	104	750
Calif.	156	115	8	270	125	110	85	3,200
Colo.	517	431	10	1,900	212	212	206	1,900
Conn.	64	55	2	70	2	2	1	75
Del.	10	10	0	8	0	–	–	–
Fla.	450	397	17	700	326	315	77	800
Ga.	170	106	1	50	10	10	5	230
Hawaii	9	9	1	100	67	65	16	200
Idaho	87	78	3	181	49	49	46	2,500
Ill.	26	26	1	340	13	11	4	100
Ind.	45	42	0	38	19	18	3	200
Iowa	33	23	4	150	5	5	4	100
Kans.	24	18	0	14	9	9	9	100
Ky.	103	97	3	80	74	69	32	1,700
La.	273	233	0	40	72	72	30	3,500
Maine	14	14	4	440	9	9	7	100
Md.	21	21	0	40	13	12	2	100
Mass.	30	29	2	124	9	9	3	1,000
Mich.	125	118	2	120	39	38	6	90
Minn.	41	33	1	50	30	29	27	900
Miss.	22	15	0	46	68	68	10	110
Mo.	74	70	10	1,000	19	19	18	550
Mont.	102	84	0	35	86	84	71	1,000
Nebr.	80	63	1	88	16	16	16	700
Nev.	13	12	0	13	10	10	10	200
N. H.	6	6	0	35	19	19	0	21
N. J.	67	66	3	240	40	40	8	100
N. Mex.	51	42	5	<2,142	63	63	63	1,600
N. Y.	203	193	2	160	248	242	60	7,800
N. C.	75	59	4	100	47	47	40	500
N. Dak.	80	72	1	100	65	65	58	500
Ohio	49	43	9	340	185	171	10	240
Okla.	55	50	1	200	61	61	61	1,200
Oreg.	25	25	1	60	13	13	13	100
Pa.	245	213	4	<21,000	207	203	179	<41,000
R. I.	11	11	0	34	4	4	4	500
S. C.	34	33	2	73	11	11	5	110
S. Dak.	45	40	0	40	17	17	11	600
Tenn.	21	19	1	50	47	45	14	300
Tex.	170	155	9	150	29	29	29	500
Utah	122	110	2	85	19	19	19	300
Vt.	4	4	1	50	1	1	0	4
Va.	17	12	0	20	4	4	1	40
Wash.	154	148	21	1,100	30	30	23	1,100
W. Va.	57	53	2	370	56	56	5	91
Wis.	38	35	0	30	14	11	2	37
Wyo.	66	45	0	15	60	59	56	600
United States	4,356	3,781	145	1,900	2,787	2,709	1,546	7,800

[a] Source: Pickering (1976).

[b] Streams, lakes reservoirs

[c] Explanation:

N = Number of stations for which data are available.
N>0 = Number of stations that have an analysis result greater than zero.
N>10μg/ℓ = Number of stations that have an analysis result greater than amount specified.
Max = Maximum value found.

Sartor and Boyd (1972) found lead to be a prevalent fraction of street dirt in 12 U. S. cities. They found that the lead accumulated over a seven-day period averaged 0.32 kg per linear kilometer (0.57 pound per curb mile). They further estimated that a storm occurring in a hypothetical city of 100,000 persons five days after the preceding storm could transport 125 kg of lead to surface streams. Erosion of soil is an analogous but much smaller source of lead. Urban soils would contribute proportionally more lead per unit mass than soils from rural areas.

Municipal wastewater may comprise another significant input of lead to surface waters. Depending on the industrial nature of the city, lead content of the wastewater can be several hundred ppb. The effectiveness of removal varies, but typically will fall in the 30-60 percent range (Brown, et al., 1973; Patterson, et al., 1975).

Areas in which lead has been mined might be expected to have greater than average amounts in the waters. The Springfield region of Missouri, which has been such a mining region, was surveyed by Proctor, et al., (1973) for lead content of surface waters and wells. In some localized areas, the lead content of the water exceeded the 50 ppb primary drinking water standard. However, most water samples had a lead content below this limit. In the Helena, Montana environmental pollution study (U. S. Environmental Protection Agency, 1972), concentrations of 1 to 40 ppb were found, which are no greater than those found in other parts of the country.

The impact of the lead mining and milling operations in Southeastern Missouri has been monitored since before the onset of development in the mid-1960's. In general, the lead (and other heavy metal) content of the streams receiving effluents have remained at or near background levels. The apparently minimum effect of these operations is attributed to the predominantly limestone geology of the area. The effect of the limestone is to produce, by carbonate precipitation, low concentrations of metals in original mine water, in mill water in tailings ponds, and even to remove any metals which reach surface waters. The local streams apparently undergo a natural purification process (with respect to lead and other metals) as they flow over limestone outcrops.

8.3.2 Lead Concentrations in Drinking Water

Most public water supplies are filtered before distribution, so that only dissolved lead is expected at the domestic tap. However, in earlier years, lead piping has been used in potable water systems, and under some conditions, high dissolved lead concentrations can be reached.

In 1969, the U. S. Public Health Service initiated an extensive community water supply survey (CWSS) which was national in scope, and included 969 public water supply systems. The samples included both surface and ground water, and large and small systems (McCabe, et al., 1970). Samples were collected at the consumer's tap rather than at the water treatment plant. Only 1.4 percent of the samples exceeded 50 ppb lead; and the highest value recorded was 640 ppb.

The U. S. Environmental Protection Agency (1976) has reported a more detailed breakdown of the distribution of lead concentrations (Table 8.9). As shown, the median concentration was about 10 ppb and only 19 of the 2,595 samples (0.7 percent) exceeded twice the primary drinking water standard.

Prior to the formation of the U. S. Environmental Protection Agency, the Public Health Service had the responsibility of certifying the water supply systems used by interstate carriers and serving the traveling public. EPA now has assumed the responsibility for monitoring the quality of drinking water utilized by interstate carriers. The most recent compilation of these results (U. S. Environmental Protection Agency, 1975f) shows that only two out of 592 analyses (0.3 percent) exceeded the 50 ppb interim primary drinking water standard.

As noted above, the contribution of lead pipes to the level of lead in drinking water can be significant. This problem is prevalent in New England because of two basic factors. First, the nature of the water supplies in New England is such that they are usually acidic and very low in naturally occurring substances such as calcium and magnesium which cause hardness, (this lead problem, accordingly, does not arise in the limestone areas of the Midwest). The second factor is the formerly widespread use of lead pipe for conveying water to homes, generally between the water main and the home; the length involved may be from 10 to 100 feet. The results of a 1974 EPA investigation of the lead levels in drinking water supplied by the Metropolitan District Commission (MDS) to the greater Boston area have been reported by Karalekas, et al., (1975). The lead concentration was less than the detection limit (13 ppb) at the treatment facility and before entering the distribution system. Based on the results of analysis of 936 samples from 383 households, it was clear that on prolonged contact with sections of lead pipe, lead content of the water reached undesirable levels. As shown by Figure 8.8, almost 50 percent of the first drawn samples in the morning exceeded the 50 ppb standard and the mean morning sample contained over 100 ppb (Figure 8.9).

To overcome this lead corrosion problem, plans were drawn up to adjust the pH of the water to reduce the aggressiveness of the water towards lead pipe; it was estimated that 2,000 metric tons (2,200 short tons) of sodium hydroxide would be required annually to accomplish this (Anonymous, 1975). Implementation of this pH adjustment accompanied by fluoridation of the water supply, began in May, 1977. The goal was to raise the pH from its former 6.5-7.2 range to 8.5. As reported by Ryan (1978), although there were the usual initial shakedown difficulties during the first several months (for example, pH varied from 7.5 to 9.2), corrosion mitigation is gradually being achieved. Evidence for this was the decrease, after several months, in the averages of the three standard grab samples (running, standing, and early morning) of the test homes from the prior range of 100 to 58 ppb, to a range of 46 to 29 ppb, below the primary water standard; and lead concentrations were still continuing to decrease.

Additional studies of five New England water systems conducted by Region I of the U. S. Environmental Protection Agency (Karelekas, et al., 1977) indi-

TABLE 8.9 LEAD CONTENT OF TAP WATER FROM TWO SURVEYS OF DISTRIBUTED WATER[a]

Concentration, µg/l	Community Water Supply Study[b]		Chicago Study[c]	
	Frequency	Percent > than stated concn.	Frequency	Percent > than stated concn.
0-1	663	100.0	205	100.0
2-3	2	74.5	18	62.7
4-5	15	74.4	13	59.5
6-7	45	73.8	13	57.1
8-9	130	72.1	24	54.7
10-14	909	67.1	97	50.4
15-19	335	32.0	17	32.7
20-24	236	19.1	49	29.6
25-29	121	10.0	55	20.7
30-34	33	5.4	7	10.7
35-39	40	4.1	24	9.5
40-44	16	2.5	3	5.1
45-49	9	11.9	19	4.5
50[d]-54	4	1.6	2	1.1
55-59	2	1.4	1	0.7
60-64	2	1.3	0	0.5
65-69	1	1.3	1	0.5
70-74	4	1.2	0	0.4
75-79	5	1.1	1	0.4
80-84	1	0.9	0	0.2
85-89	1	0.8	0	0.2
90-94	0	0.8	0	0.2
100-199	2	0.8	1	0.2
200-299	12	0.7		
300-399	2	0.3		
400-499	3	0.2		
500-	2	0.1		
	2595		550	

---

[a] Source: U.S. Environmental Protection Agency,(1976)

[b] Data from McCabe, et al., (1970)

[c] Data from Mc Cabe and Vaughn (1969)

[d] Primary drinking water standard.

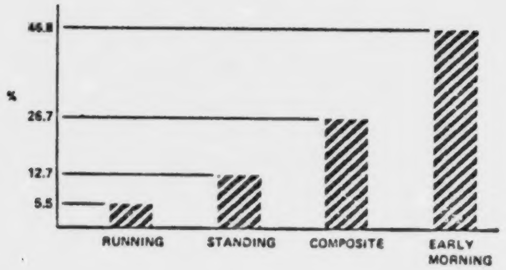


Figure 8.8 Percentage of Boston tapwater samples exceeding lead standard

Source: Karelekas, et al., (1975).

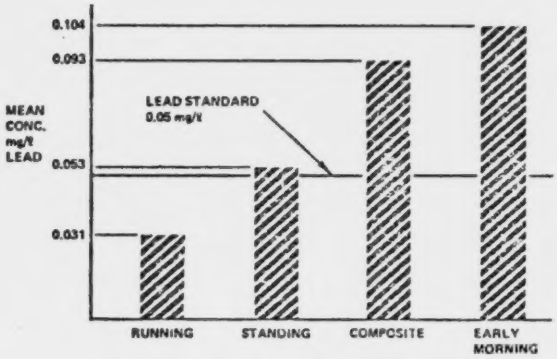


Figure 8.9 Mean lead concentration of Boston tap water samples by type of sample.

Source: Karelekas, et al., (1975).

cated that the adjustment of pH alone to the range of 7.0 to 8.0 did not reduce the lead concentrations below the standard, and that at pH's below 7, the use of phosphate corrosion inhibitors (e.g. zinc orthophosphate, sodium zinc phosphate, and sodium hexametaphosphate) did not eliminate above-standard concentrations. The 3 systems with above-standard lead concentrations characteristically had very soft water, with hardness values in the 10 to 20 ppm range, along with a pH below 7.5. In contrast, the 2 systems which did not exhibit above-standard lead concentrations had hardnesses of 40 ppm or higher, and a pH above 7.1 (in one case, a pH of 10.1). Both of these systems adjusted pH with lime, which also adds calcium hardness.

While these data are somewhat limited, the indications are clear that control of lead concentrations to below primary water standards can be achieved with relatively simple adjustments of water pH and composition.

Lead pipe is no longer used for potable water systems in this country, and its use is contrary to most current building codes. Most new domestic water piping systems employ copper pipe, which is generally joined (sweated) with either a 50:50 or 40:60 lead: tin solder (see Section 4.3.5.2). Surprisingly, there has been almost no research on lead pickup from such systems; only one study has been identified (Wong and Berrang, 1976), and it is so briefly described that intrepretation and evaluation is difficult. This study concerned some simulated systems exposed to tap water from the Victoria, B. C., Canada supply. Characteristics of the water were not disclosed, but it is known to be similar to that of Boston, low in pH and dissolved minerals, and quite aggressive. This study indicated that the 50 ppb limit could be exceeded in a system with 20 soldered joints after one hour's standing, depending on the amount of water which had been passed through the system. Tests on a real system in a one-year-old house yielded a dissolution rate of 0.4 μg/Pb/joint/hr. with lead service pipe dissolution rates were 30-480 μg/hr. Evaluation of the importance of solder joints on lead exposure will have to await the conduct of some more definitive and better documented investigations.

8.3.3 Trends in Water Lead Concentrations

There are no indications that lead concentrations of U. S. surface waters are increasing; and ground water has always been low overall. Two principal reasons for this can be hypothesized; the low solubility of lead in most natural waters; and the input of lead to the environment is diminishing as lead antiknock usage declines, and as compliance with Federal air and water emission regulations increases. The promulgation of new pretreatment guidelines will reduce lead inputs to municipal wastewater treatment plants, which will have beneficial effects on downstream lead concentrations.

Recognition of lead problems at the consumers tap in a few geographical areas is well advanced. Methods of minimizing lead corrosion are available, and it is anticipated that these problems will be overcome in the near future.

As these mitigating procedures are implemented, a gradual decrease in the mean lead content of U. S. water should become evident.

8.4 LEAD IN SOILS

In terms of occurrence in the earth's crust, lead is a rare metal, amounting to only a fraction of such other metals as copper, zinc, and iron. Earlier work on measurement of lead in soils and rocks has been summarized by de Treville (1964) who reported data which were compiled mainly at the Kettering Laboratory. These data indicate that an average value for the earth's crust is 16 ppm, with alkaline rocks containing about 8 ppm and acidic rocks about 20 ppm.

8.4.1 Soil Lead Concentrations in the U. S.

The U. S. Geological Survey has recently conducted an extensive survey of the elemental composition of surficial materials in the conterminous United States (Shacklette, et al., 1971). These results, summarized and updated with new analyses by Tidball (1976), indicated that the moister soils of the eastern half of the country had a mean lead concentration of 14 ppm, as compared with 18 ppm for the drier soils of the west. About 94 percent of the 964 samples had lead concentrations of 30 ppm or below, and about 58 percent had concentrations equal to or less than the mean of 16 ppm.

Comparison of the lead content of urban soils in a region which included industrial, agricultural, and residential areas showed that there was about 2.7 times as much lead in industrial soil as in residential soil (Klein, 1972). The agricultural soils contained slightly less lead than those in residential areas.

8.4.2 Localized Source - Oriented Studies

Numerous studies have been conducted to determine the effect of various types of lead sources on the close-in deposition on soils. Not surprisingly, automobile and highway-oriented studies lead the list, followed by smelter-oriented investigations; a relatively limited number of studies have been conducted to determine the effects of dispersion of lead from painted houses, and of the cumulative accumulation in orchards from the application of lead arsenate insecticides.

Several studies illustrative of the highway-related investigations are noted below and briefly summarized; details can be found in the original reference. Lagerwerff and Specht (1970) present data typical of the depth profiles for lead found by various investigators. Table 8.10 summarizes the results, and shows about a 65 to 75 percent reduction in lead content with distance in the top 5 centimeters of soil for samples taken at points 8 to 32 meters from a highway. In every case, the amount of lead decreased with depth of sample.

The accumulation of lead in soils over a period of about 40 years was compared for areas of high and low motor vehicle traffic densities in the Los Angeles area (Page and Ganje, 1970). The sampling sites were more than 1.6 kilometers from any major highway. No lead accumulations as a function of time were observed where motor vehicle traffic was less than 80 vehicles/2.6 km^2 ($80/mi^2$). In areas where the traffic density exceeded 580 $vehicles/mi^2$, the surface concentration of lead (2.5-cm depth) increased by a factor of 2 to 3 during the 40 year period, which amounted to an accumulation of 15 to 36 ppm of lead. The highest value recorded for any location was 52 ppm.

A very high lead concentration was found near a heavily-travelled expressway in Chicago. The soil contained as much as 7,600 ppm at distances up to 13.7 meters from the expressway, and 900 ppm up to 45.7 meters (Khan, et al.,

Table 8.10 LEAD CONTENT IN ROADSIDE SOIL AND GRASS AS A FUNCTION OF DISTANCE FROM TRAFFIC AND SOIL DEPTH[a]

Site	Distance from Road, m	Lead Content, μg/g dry weight			
		Grass	Soil Profile Layer, 0-5 cm	Soil Profile Layer, 5-10 cm	Soil Profile Layer, 10-15 cm
I	8	68.2	522	460	416
West of U.S. 1, near Plant	16	47.5	378	260	104
Industry Station, Beltsville, Maryland	32	26.3	164	108	69
II	8	51.3	540	300	98
West of southbound lanes,	16	30.0	202	105	60
Washington-Baltimore Parkway, Bladensburg, Maryland	32	18.5	140	60	38
III	8	21.3	242	112	95
West of Interstate 29,	16	12.5	140	104	66
Platte City, Missouri	32	7.5	61	55	60
IV	8	31.3	150	29	11
North of Seymour Road,	16	26.0	101	14	8.2
Cincinnati, Ohio	32	7.6	55	10	6.1

[a] Source: Lagerwerff and Specht. Reprinted with permission from Environmental Science and Technology. (c) American Chemical Society. (1970).

1973). The amount of lead varied with the seasons in a fashion similar to the seasonal variations in average monthly traffic volumes on the expressway. Lead levels were lowest during fall and winter, increased during the spring, and reached their highest values during the summer.

Hamphill, et al., (1974) studied roadside lead contamination in soils along highways used for transport of lead concentrate in the Missouri lead belt. Lead values approaching 3,800 μg/g (ppm) of dry weight were found at zero distance from the road. Along control routes, high lead values of 384 μg/g of dry weight were observed. The total lead in soils decreased rapidly at 100 to 300 meters. The investigators suggested that the elevated levels of lead in the soil near the control routes are probably due to contamination from motor fuel and possibly other sources.

The presence of a lead smelter can cause very high lead levels in nearby soils. In the Helena, Montana study cited previously, the upper 2.5 centimeters of uncultivated soil were found to contain 4000, 600, and 100 ppm of lead at distances from the smelter of 1.6, 3.2, and 6.4 kilometers (1, 2, and 4 miles), respectively (U. S. Environmental Protection Agency, 1972).

Roberts, et al., (1974) found that a high rate of lead fallout around two secondary lead smelters originated mainly from large-particulate emissions from low level fugitive sources rather than from stack fumes. The lead content of dustfall and, consequently, of soil, vegetation, and outdoor dust, decreased exponentially with distance from the two smelters. Lead emissions from the two smelters were estimated to be 15 to 30 metric tons per year. Regression analysis of the concentrations indicated an exponential decrease with distance, from values of 40,000 and 16,000 μg/g of soil close to smelter A and smelter B, respectively, to an urban background of 100 to 500 micrograms per gram of soil, which accounted for 60 to 80 percent of the variability in data. Although the monthly geometric means of the atmospheric lead concentrations close to the smelters (1 to 5.3 $\mu g/m^3$ of air) were only double those for urban sites (0.8 to 2.4 $\mu g/m^3$) the range of the daily concentrations was much greater, producing a marked log normal distribution.

As a part of the environmental pollution investigation at a smelter in El Paso, Texas (see Section 8.2.2.2) soil samples were also collected at 99 sites in El Paso and at three remote sites. Samples were taken at the surface and at depths of 2.5, 5.0 and 7.5 cm (1, 2 and 3 inches). Only trace amounts of lead were found (<50 ppm) at the remote sites. Within the city, highest levels in 1972 were found within 200 m of the smelter (mean of 3457 ppm and range of 560 to 11,450 ppm for 54 samples); lead content was consistently highest at the surface. Concentrations fell rapidly over the first 2 to 3 km from the smelter, but remained above background for as far as 10 km (Landrigan, et al., 1975).

The use of lead paints on the exterior of houses has been found to result in locally high concentrations of lead in soils. Soil samples taken near old houses in Cincinnati built prior to 1900 had lead concentrations which ranged from 32.5 to 7,620 ppm. In general, there was a correlation between soil lead, and the lead content of the exterior paint on the houses (Bertinuson and Clark, 1973). This work also demonstrated that much more lead could be accumulated in the soil around a house with lead paint than at the roadside where heavy automobile traffic passed (10,200 vehicles per day). The house contribution to soil lead was about 30 times greater than that from the road. This source of lead can be accentuated if the old paint is chipped and scraped from the wood and then allowed to disperse in the vicinity. One such measurement showed that lead in the soil 15 to 30 meters from the house was 165 to 185 ppm when the old paint was removed. A year later, the lead content had increased to 440 to 490 ppm (Bogden and Louris, 1975).

It has long been recognized that the eating of lead paint is a main cause of lead poisoning and elevated blood leads of children living in dilapidated housing. In this connection, Ter Haar and Aronow (1974) determined the source of lead in dirt to which children are normally exposed. Dirt samples were taken in old urban areas around 18 painted frame houses and 18 houses of brick construction (Table 8.11). The data suggested that (1) weathered lead-based paint is a major contributor to soil lead, and (2) vehicular sources probably make a significant contribution to soil lead near the sidewalks.

High local concentrations of lead might be expected to occur in soils where plants and trees have been sprayed with lead arsenate. In past years, this compound was used as an insecticide but has been presently supplanted by organic compounds. However, the soil of an orchard which had been sprayed with lead arsenate for many years was found to contain about 50 to 65 ppm of lead. This is not an unusually high value; however, it is significantly greater than the 15 to 20 ppm which is typical of uncontaminated areas (Vandecaveye, et al., 1936).

8.4.3 Lead in Urban Dusts

Street dust and house dust can also be a source of lead, especially for young children prone to hand-to-mouth activity. In a survey of 77 midwestern cities, it was found that the average lead concentration in the street dust of residential areas was 1658 ppm, and that in commercial and industrial areas the average concentrations were 2,334 and 1,386 ppm, respectively (Hunt, et al., 1971).

Day, et al., (1975) analyzed several hundred samples of urban dust and dirt likely to be encountered by children as a normal part of their environment in Manchester, England, with results in general agreement with those of Hunt, et al. The average lead concentration in the urban samples was 970 ppm (median concentration was 860 ppm). The highest value encountered was 10,200 ppm, and the lowest 90 ppm. The higher values were unusual; only 10 percent of the samples exceeded 1,500 ppm, and only 5 percent exceed 2,000 ppm.

Solomon and Natusch (1977) conducted an area-wide survey of settled dusts and soils in a moderate-sized urban community (Urbana-Champaign, Illinois). Approximately 1400 samples, both indoor and outdoor, were taken in residential and nonresidential areas. The homes selected were in good repair, painted with

TABLE 8.11 LEAD IN DIRT IN DETROIT[a]

Location	Lead Concentration, ppm			
	Painted Frame Houses		Brick Houses	
	Mean	Range	Mean	Range
Within 0.6 m of house				
front	2349	(126-17590)	351	(78-1030)
back	1586	(162-4951)	501	(72-2350)
sides	2257	(140-7284)	426	(91-1160)
3 m from house				
front	447	(58-1530)	156	(39-316)
back	425	(149-1410)	200	(72-480)
Near sidewalk	627	(152-1958)	324	(86-1130)
curb	572	(320-1957)	612	(147-2420)
Gutter	966	(415-1827)	1213	(304-3170)

[a] Adopted from Ter Haar and Aronow (1974).

unleaded or low-lead paints, located in relatively low traffic density areas, and were described as upper-middle-class abodes in extremely good condition. The average lead content of 239 floor dust samples taken in 12 homes was 600 ppm (680 μg/m^2 floor surface).

Lead was also determined in 228 outdoor soil and dust samples around the same houses. Lead levels in dusts were high both near roads and near the houses. The range in dust lead content was from 240 to 6640 ppm away from the houses and from 130 to 11,760 ppm near them. Soil lead was lower and less variable, 20 to 1060 ppm.

In 27 house dust samples collected in the adjacent and far suburbs of the Boston and Cambridge, Massachusetts area (Kreuger, 1972, cited in Solomon and Natusch), lead concentrations of 1,200 ppm were found. Lead concentrations in house-dust samples collected in the core city averaged 2,000 ppm; core-city gutter samples ranged from 400 to 9,000 ppm. Lead levels measured in these studies are much lower than those usually found in truly dilapidated homes -- 50,000 to 400,000 ppm (National Academy of Sciences, 1972).

Illustrative of the latter type of situation is the investigation of Lepow, et al., (1975) in a deprived area of Hartford, Connecticut, to ascertain the cause of the chronically elevated blood levels (> 40 μg/dl) in ten 2-6 year old children. The mean age was 52 months, and the blood levels were 40-70 µg/dl. Several of the children had reached peak blood lead levels of 70-120 µg/dl, and half had received at least one course of chelation therapy. All but one had a previous history of pica, and 9 of the 10 study children exhibited some degree of excessive mouthing behavior.

The average lead concentration in outdoor play area dirt was 1,200 ppm (range 700-1,750 ppm), and the average in house dust was 11,000 ppm (range 4,900-17,000 ppm). Dirt sampled from the hands of the study children averaged 2,400 ppm lead.

These averages from Hartford should not be regarded as typical, even for urban environments. They do illustrate the dimensions of the problem in combatting the lead intoxication problem found in economically deprived children living in old and deteriorated housing.

8.4.4 Trends in Soil Lead Concentrations

The mobility and persistence of anthropogenic lead in soil is influenced by its chemical form. The nature of the lead compounds in soil that originate in automobile exhausts and that are formed near roadways was determined by Olson and Skogerboe (1975). Samples were collected from the top centimeter of surface soil or from street dust in four different parts of the U. S. Lead sulfate accounted for the major portion of the lead present in the samples. A small amount of $PbO \cdot PbSO_4$ and traces of lead oxides were found in some of the specimens, while PbS was detected in a sample from the Missouri Lead Belt. In view of the work by Ter Haar and Bayard (1971) which showed that only about 3 percent of the aerosol lead in "old" exhaust particulates was $PbSO_4$, it appears that much of the conversion to lead sulfate occurs after the lead compounds are deposited on the soil.

If the lead is of strictly inorganic origin, it apparently can be leached from the soil at a fairly rapid rate. At the sites of permanently shutdown smelters, it was found that badly contaminated surface soil (5-cm) (104 to 328 µg/g) lost lead at a rate that would reduce it to background values in 2 to 5 years (see Section 6.6.1).

In general, lead deposited on the soil is retained near the surface, and its concentration drops off rapidly with depth of the soil (e.g. see Table 8.10). In most respects the soil can be considered as a near-total sink for lead received by it. Thus, its lead content can be expected to increase with time, although at an almost imperceptible rate except where emissions from local point sources are large and concentrated.

If lead from the combustion of leaded gasolines is the major contributor to the lead content of urban dusts, as seems likely from the available data, the gasoline lead phasedown should result in proportional decreases in dust lead contents. A concentration decrease of 2/3 or greater by 1980 from the 1970 peak would be compatible with the phasedown schedule.

8.5 LEAD IN FOODS AND BEVERAGES

This section presents data on natural or "background" levels of lead in foods, sources and mechanisms of lead uptake in foods and discusses the findings of numerous investigations regarding the lead content in various types of foods. Interpretation of the potential human health impact of these findings, typical dietary dosages (human exposure patterns) and problem areas are discussed in Sections 8.6 and 8.7.

Lead is naturally present in foods because it is a normal constituent of soils (averaging 16 ppm in the United States). The quantity of lead derived from soils (unless contaminated) is generally low; of the order of a few tenth of 1 ppm (Kehoe, 1961). Kehoe's early investigations (Kehoe, et al., 1933) revealed lead in every food item both in the field and from dwellings of the inhabitants of a primitive region unexposed to industrial or mining activities. Patterson (1965) estimated that the natural lead content of food should be 0.01 µg/g wet weight or 0.01 ppm. Because the lead content of most foods today is much higher than 0.01 ppm, this author concluded that most of the lead present in today's foods arises from industrial sources of lead. National Academy of Science (1972) cites the work of Schroeder, et al., (1961) who found approximate means and ranges as follows:

	Lead Content, wet weight, ppm	
Commodity	Mean	Range
Condiments	1.2 ppm	0 - 1.5
Fish and seafood	0.5 ppm	0.2 - 2.5
Meat and eggs	0.2 ppm	0 - 0.37
Grains	0.4 ppm	0 - 1.39
Vegetables	0.2 ppm	0 - 1.3
Fresh whole milk	No detectable lead	

The NAS document (National Academy of Sciences, 1972) cites evidence to suggest that the average lead content in foods is about 0.2 ppm.

Soil, dustfall, and rainfall are potential sources of lead in plants which are related, to some degree, to ambient air conditions. Other known sources of lead in foods are: (1) usage (formerly) of lead arsenate pesticides, (2) surface deposition of lead near roadways, (3) inadvertent addition of lead during food processing or canning, (4) leaching from improperly glazed utensils or storage vessels and, possibly, leaching from some types of plastic containers.

8.5.1 Determinants of Lead Content

The lead content of food or forage plants is related to the amount of lead applied topically to exposed areas of edible leaves, stems, and fruit by rainfall or dustfall, and to the amount that is translocated from soil and surface and/or ground waters through the roots to stems, leaves, and fruit (Bethea and Bethea, 1975; Ewing and Pearson, 1974; Kehoe et al., 1968; Haley, 1969; Leland et al., 1975; see Section 6.6.2). Lead may accumulate differentially in different portions of plants, in varying quantities depending upon weather, proximity to highways, traffic patterns, etc. (Leland et al., 1975; Schuck, 1970; Schuck and Locke, 1970). The lead content of portions that are eaten by humans or livestock are most important from the health standpoint (Haley, 1969; International Lead Zinc Research Organization, 1972; Kolbye, et al., 1974).

Lead appears to accumulate differentially in various portions of the plant. In general, lead concentrations tend to be low in the fruit grain and tubers (except for seeds) and higher in the roots and leaves (Motto, et al., 1970). Topical application of lead from airborne fallout is usually of minimal significance to humans because the edible portions tend to be protected by coverings and because 1/2 - 2/3 of the lead deposited in this fashion washes off. Since food for human consumption is normally washed before eating, the contribution of topical lead is probably small. Note, however, that animals consuming these commodities would not be similarly protected.

For meats, milk, eggs and other products from domestic livestock, the lead content is related to that of the feed, forage and water consumed by the animals (Engel, et al., 1971; Ewing and Pearson, 1974; Guss, 1970; Lynch, et al., 1974). Animals grazing near roadways or stationary sources of lead emissions may be vulnerable to lead intake from heavily contaminated grasses and vegetation. Meats consumed by humans conceivably could be contaminated in this manner, yet, the National Academy of Sciences (1972) reports that there is no evidence that this is occurring. The lack of measurable changes in lead content of meats and other animal food products over the past 30 years also supports this contention.

In the case of fish and shellfish, lead content in tissues is related to that of the aquatic (fresh, brackish or marine) environment and to the uptake of lead by organisms such as plankton in the food chain (Dorn, et al., 1972; Leland, et al., 1975; Loutit, et al., 1973).

In addition to the lead derived from air, aoil, and water, foods may contain lead derived from glazed vessels and metal containers sealed with lead solder (Clark, 1972; Harris and Elsea, 1967; Klein and Namer, 1970; Mitchell and Aldous, 1974). Lead poisoning has occurred from the consumption of homemade apple cider made in lead-lined earthenware vessels and illegal alcohol or "moonshine" produced in old automobile radiators in the southwestern states (Palmisano, et al., 1969; Walls, 1969).

8.5.2 Lead Content of Individual Food Commodities

8.5.2.1 Food and Forage Plants--

A substantial body of published data exists on the lead content of food and forage crops, including cereals, vegetables and fruits. Tables 8.12 and 8.13 summarize typical values for the lead content of cereals and vegetables, and of fruits, respectively.

The available data on portions of crop plants whose lead content is derived from both air and soil indicate that soil uptake is the more important source. Lead from the air generally deposits on the leaves and outer portions of edible plants such as husks, as well as on the soil. It appears that uptake of lead through the stomata of leaves may be considerably less than that taken up from soil through the roots. However, crops grown close to highways (within 30.4 meters or 100 feet) having a heavy traffic volume may have high levels of lead contamination on the leaves and outer portions. The question of whether or not lead is translocated from the leaves or roots to other portions of crops has not been resolved (see Section 6.6.2). The available data indicate that the edible portions of crop plants including leaves, roots and fruits generally have a lower lead content than inedible portions. Data obtained by the Food and Drug Administration from analyses of "market basket" samples of food purchased in retail establishments indicate that levels of lead consumed in fresh fruits and vegetables are well below levels considered significant from the human health standpoint (Kolbye, et al., 1974).

Leafy wastes from food crops that contain high levels of lead may be a hazard to domestic livestock if used as a component of feed. For example, note in Table 8.12 the considerably higher lead content of corn stalks, leaves, and husks as compared to the kernels, and of unharvested cabbage leaves as compared to the head. The type of lead poisoning in cattle and horses is more fully discussed in Section 6.5.2.

Possible hazards from lead contamination of homegrown vegetables has only recently been considered. A warning, for example, was recently issued by the Idaho Department of Health and Welfare to residents within a 6.4-km (4-mile) radius of a lead smelter that homegrown vegetables may have high levels of lead (Anon, 1975). It is likely, however, that vegetable contamination in smelter areas is primarily a problem of deposition of lead, particularly on the external portions of the plants rather than high concentrations of lead in the plant tissues themselves. Thus, thorough washing should remove most of the lead deposited on the surface. In any event, unless the garden provides a substantial proportion of the total diet, the effects of lead contribution from contaminated produce should be diluted in the total pool of food consumed so that this source probably would contribute very little total lead in a well balanced diet.

TABLE 8.12 LEAD CONTENT OF CEREALS AND VEGETABLES

Products	Lead Content, ppm	Sample State	Reference
Wheat, heads	1.0-2.0	Dry	(1)
grain	0.16-0.18	Dry	(2)
Oats, heads	0.3-2.0	Dry	(1)
Corn, tassels	19-1742	Dry	(3)
leaves	30-329	Dry	(3)
stalks	3.1-13	Dry	(3)
kernels	0-2.6	Dry	(3)
cobs	0.9-5.6	Dry	(3)
Barley, heads	0.5-2.0	Dry	(1)
Rye, heads	0.3-7.0	Dry	(1)
grass	2.4-14.2	Dry	(4)
Cabbage, heads	1.0-1.1	Dry	(2)
	<0.01-0.51	Wet	(5)
unharvested leaves	4.5-5.8	Dry	(2)
Cauliflower, hearts	0.9	Dry	(1)
leaves	2.0	Dry	(1)
Broccoli, flowers	0.19-0.30	Wet	(6)
stalks	0.04	Wet	(6)
Brussel sprouts	<0.01-0.24	Wet	(5)
Collards	0.25-0.50	Wet	(6)
Celery	<0.01-0.02	Wet	(6)
	0.01-0.02	Wet	(5)
Spinarch	0.10-0.25	Wet	(6)
Watercress	0.01-0.33	Wet	(5)
Lettuce, leaves	8.7-159	Dry	(3)
	3.2-6.6	Dry	(2)
	1.0-9.0	Dry	(1)
roots	20-150	Dry	(3)
Lettuce, Romaine, outer leaves	0.10-0.18	Dry	(6)
inner leaves	<0.01-0.01	Wet	(6)
Artichokes, outer leaves	0.04	Wet	(6)
inner leaves	<0.01	Wet	(6)
Potatoes, leaves	20-530	Dry	(3)
stems	14-53	Dry	(3)
roots	7.6-100	Dry	(3)
tubers	1.2-14	Dry	(2)
	0.30-0.33	Dry	(2)
	<0.01-0.14	Wet	(5)

TABLE 8.12 LEAD CONTENT OF CEREALS AND VEGETABLES[a]
(Continued)

Products	Lead Content, ppm	Sample State	Reference
Potatoes, tubers, peel	7.4-10.4	Dry	(3)
tubers, inner portion	0.02	Wet	(6)
	3.3-4.1	Dry	(3)
Onions	<0.01-0.38	Wet	(5)
Beets, roots	1.6-6.0	Unspecified	(7)
	1.0-6.0	Dry	(1)
tops	2.0-10.0	Dry	(1)
Carrots, tops	25-218	Dry	(3)
	4.70-7.0	Dry	(1)
leaves	83-152	Dry	(3)
stems	16-23	Dry	(3)
roots	0.05-0.06	Wet	(6)
	3.3-24	Dry	(3)
	1.7-2.1	Dry	(2)
		Wet	(5)
	2.0	Dry	(1)
inner portion	<0.01	Wet	(6)
Leeks	0.04-0.07	Wet	(5)
Radish, leaves	2.3-16.4	Dry	(4)
roots	0.8-2.0	Dry	(4)
	0-0.2	Unspecified	(7)
Peas, pods and peas	<0.01-0.02	Wet	(6)
pods	<0.01-0.02	Wet	(6)
peas	0.03	Unspecified	(7)
Beans	0.15-0.26	Unspecified	(7)
	1.2-1.4	Dry	(2)
Beans, leaves	7.9-20.0	Dry	(2)
Cucumbers	0.01-0.03	Wet	(5)
Tomatoes, leaves	37-276	Dry	(3)
blades	31-87	Dry	(3)
petioles	8-16	Dry	(3)
stems	3.9-166	Dry	(3)
fruit	0.59-0.72	Dry	(2)
	0.01-0.14	Wet	(5)
peels	3.6-16	Dry	(3)
pulp	3.1-6.8	Dry	(3)
Rhubarb, stems	2.0-4.0	Dry	(1)
leaves	8.0-11.0	Dry	(1)
Mushrooms	0.03-0.04	Wet	(5)

[a] References:
(1) Warren and Delavault (1962)
(2) Ter Haar (1970)
(3) Motto, et al., (1970)
(4) Dedolph, et al., (1970)
(5) Thomas, et al., (1972)
(6) ILZRO (1972)
(7) de Treville (1964)

TABLE 8.13 LEAD CONTENT OF FRUITS

Products	Lead Content, ppm	Sample State	References[a]
Apples, whole fruit	0.12-0.3	Unspecified	(1)
	<0.01-0.37	Wet	(2)
skin	<0.01-0.24	Wet	(2)
flesh	<0.01-0.05	Wet	(2)
Pears, whole fruit	0.18	Unspecified	(1)
	0.02-0.04	Wet	(2)
skin	<0.01-0.23	Wet	(2)
flesh	<0.01-0.03	Wet	(2)
Peaches	0.04-0.08	Unspecified	(1)
Cherries	0.12	Unspecified	(1)
Plums	0.06-0.16	Wet	(3)

[a] References: (1) de Treville (1964). (2) Thomas, et al., (1972). (3) Thomas et al., (1973).

8.5.2.2 Processed Fruit and Vegetable Products--

Table 8.14 summarizes reports of analyses of lead contamination in canned, frozen, and dehydrated fruit and vegetable products. The lead content of frozen beans, peas and carrots was below the detectable limit of the analytical method used. Interestingly, these products, together with frozen spinach and frozen potatoes, had a lower lead content than that of the corresponding fresh products given in Table 8.3. The canned fruit products listed in Table 8.14 had significantly higher lead contents (99.9 percent confidence level) than the corresponding fresh products listed in Table 8.13.

Various studies have provided convincing evidence that the differences between fresh and canned products result from the release of lead from solder used along the seam of the can or possibly from inadvertent addition of lead during other phases of processing operations. These studies are discussed in detail in Section 8.6.4.2.

TABLE 8.14 LEAD CONTENT OF PROCESSED FRUITS AND VEGETABLE PRODUCTS[a]

Products	Lead Content, ppm	Sample State	References[a]
Beans, frozen	<0.01	Wet	(1)
Beans, baked	0.19-0.67	Wet	(2)
Carrots, frozen	<0.01	Wet	(1)
Corn, canned	0.00-3.00	Wet	(3)
Peas, frozen	<0.01	Wet	(1)
Peas, canned	0.00-1.46	Wet	(3)
Spinach, canned	0.13-0.95	Wet	(2)
Spinach, frozen	0.07	Wet	(1)
Potatoes, frozen	0.01	Wet	(1)
Tomatoes, canned	0.32-0.63	Wet	(2)
Apples, canned	0.12-1.14	Wet	(2)
Apricots, canned	0.52-1.48	Wet	(2)
Apricots, dried	0.24	Unspecified	(4)
Black currants, canned	0.17-3.90	Wet	(2)
Damsons, canned	0.37-0.74	Wet	(2)
Grapefruits, canned	0.19-0.54	Wet	(2)
Oranges, canned	0.10-0.22	Wet	(2)
Peaches, canned	0.26-0.50	Wet	(2)
Pineapples, canned	0.14-0.54	Wet	(2)
Plums, canned	0.17-0.98	Wet	(2)
Prunes, canned	0.15-0.73	Wet	(2)
Raisins	0	Unspecified	(4)
Rhubarb, canned	0.10-2.57	Wet	(2)
Strawberries, frozen	0.02	Wet	(1)
Fruits and vegetables, dried	1.0-2.0	Unspecified	(4)

[a] References:
(1) ILZRO (1972a)
(2) Thomas, et al. (1973)
(3) National Food Processors Association (1977)
(4) de Treville (1964)

8.5.2.3 Meat, Fish and Poultry Products--

Table 8.15 presents a summary of the lead content of meat, fish and poultry products. The lead content of organ meats such as heart, kidney and liver of swine and turkeys (not shown) is usually higher than that of muscle (Dalton and Malanoski, 1969). However, the lead content of beef liver appears to be

TABLE 8.15 LEAD CONTENT OF MEAT, FISH, AND POULTRY PRODUCTS[a]

Products	Lead Content, ppm	References[c]
Beef, roast chuck	0.71[b]	(1)
bone of leg	3.60	(2)
hamburger	0.248[b]	(1)
liver	0.089[b]	(1)
Pork, fresh, muscle	0.54-1.33	(3)
Meat, canned, cured[a]	0.01-0.30	(4)
wieners (smoked or unsmoked)	0.04-0.16	(4)
bologna	0.01-0.26	(4)
meat loaves	0.01-0.26	(4)
salami	0.01-0.30	(4)
pastrami and other	0.01-042	(4)
liverwurst	1.80-7.60	(5)
bacon	0.01-0.16	(4)
ham	0.01-0.16	(4)
picnic shoulders	0.01-0.12	(4)
corned beef products	0.01-0.28	(4)
Fish, fresh		
cod, fresh, Icelandic	0.32	(6)
clams, hard, Atlantic	0.28	(6)
crab, body meat, Pacific	0.45	(6)
lobster, Atlantic	0.55	(6)
shrimp, Alaskan	0.50	(6)
Fish, canned		
tuna, initial	0.34	(7)
canned 6 months after packing	0.44	(7)
sardines, Dutch	0.72	(8)
Poultry		
chicken, fryer	0.127[b]	(1)
turkey, fresh, muscle	2.93-3.35	(3)
Eggs, chicken	0.174[b]	(1)
Meat, fish, and poultry (composite)	0.015	(9)

[a] Data on canned, cured meats are expressed on a wet-weight basis.

[b] Mean.

[c] References:
(1) U.S. Department HEW (1975)
(2) Kehoe (1976)
(3) Dalton and Malonaski (1969)
(4) Kirkpatrick and Coffin (1973)
(5) Hankin, et al., (1975)
(6) Zook, et al., (1976)
(7) National Food Processors Assn. (1977)
(8) Reith, et al., (1974)
(9) Kolbye, et al., (1974)

quite low compared to that of beef muscle.

The lead content for cured and smoked meat products given in Table 8.15 was reported by Kirkpatrick and Coffin (1973) on a fresh-weight basis. When these are corrected to a dry-weight basis, the highest lead level of bacon, ham and picnic shoulders will be lower than that for pork muscle reported by Dalton and Malanoski (1969). According to Hankin, et al., (1975), seven market samples of fresh beef and pork liver that were analyzed by the atomic absorption spectrometric method of Dalton and Malanoski (1969) contained 1.4 to 1.6 ppm Pb. Liverwurst sampled sporadically contained from 0 to 7.6 ppm Pb, apparently depending upon the lead content of the ingredients used.

Fish reportedly contain lead in the range of 0.1-1.0 ppm. This is at least 2-3 order of magnitude higher than the water from which they are harvested but is more nearly comparable to the concentrations in the flora and fauna utilized as food by the fish (Ewing and Pearson, 1974). The National Marine Fisheries survey of selected seafoods reported mean lead values among species which tended to be rather uniform and only a few species averaged higher than 0.6 ppm. No species exceeded 1.0 ppm (Zook, et al., 1976). Unfortunately, many data on lead in fish (i.e., wildlife surveys) report on a whole fish basis including internal organs. "Edible portions" analyses, relevant for humans, are reported much less frequently. FDA's Market Basket studies composite fish with meats and poultry and, therefore, no average values for fish can be derived from that data.

8.5.2.4 Milk and Infant Formulas--

There is considerable concern about the lead content of milk and processed milk products since these foods comprise a substantial proportion of the diet of infants and children. Various data, past and present, exist with respect to lead levels in milk, processed milk products, infant formulas and human milk. These data are assembled by product in chronological order in Table 8.16.

Numerous articles in recent years have discussed the difficulty of accurately quantifying lead in milk in the parts per billion range (Lamm and Rosen, 1974; Brandt and Bentz, 1971; Kolbye, et al., 1974, and Dutilh and Das, 1971). Differences as large as tenfold between different investigators and/or analytical techniques in analysis of the same product are not uncommon. Cooperative studies involving several laboratories using standardized methods of sample preparation and analytic techniques on identical samples have revealed poor performance and extreme variability between laboratories. Accuracy becomes worse as the level of lead decreases, particularly as the lead level decreases below 1 ppm, which is typical of milk products. In addition, the ubiquity of lead magnifies the possibility for sample contamination. On the other hand, trace metal loss during destruction of the sample matrix also contributes to error. These problems notwithstanding, several generalizations can be made from the data shown in Table 8.16.

TABLE 8.16 LEAD CONTENT OF MILK AND INFANT FORMULAS

Milk Product	Year of Sampling	Lead Content, ppm		Analytical Method[d]	Reference[f]
		Mean	Range		
Canned evaporated milk					
skimmed	1971	1.04	0.30-2.30	AAS[d]	(1)
	1972-73	0.06	0.04-0.07	AAS	(2)
regular	1968	0.81[c]		AAS	(3)
	1971	0.36	0.30-0.40		(1)
	1972-73	0.11	0.04-0.22	NAAS	(2)
	1972-74	0.14	0.03-0.91	undetermined	(7)
	1973	0.202	0.01-0.820	AAS	(4)
	1973-74	0.125	0.02-0.37		(5)
		0.12	0.01-0.46		(11)
Infant Formula					
Concentrate	1972-73	0.083	0.04-0.12	NAAS	(2)
		0.09	0.06-0.13	AAS	(6)
Ready to serve (diluted)					
Modified Milk I	1968	0.170		AAS	(3)
Modified Milk II	1968	0.210		AAS	(3)
Lamb Meat Base	1968	0.256		AAS	(3)
Soya Base	1968	0.272		AAS	(3)
	1972-73	0.033[b]	0.-0.08	NAAS	(2)
	1972-74	0.05		undertermined	(7)
Homogenized Milk (cow)	1971	0.04		AAS	(1)
	1972-73	0.005[a]		NAAS	(2)
	1973	0.04		AAS	(4)
	1972-74	0.02	0.02-0.07	not stated	(7)
		0.005		dithizone	(8)
		0.042[e]	0.009-0.1540	AAS	(9)
		0.042		photon activation	(10)
		0.049	0.023-0.079		
Human Breast Milk					
	1971	0.02	0. -0.065	AAS	(1)
	1972-73	0.005		NAAS	(2)
		0.019[e]	0.006-0.202	AAS	(3)
		0.012		AAS	(3)

[a] Detection limit 0.005 ppm.
[b] Seamed cans and bottles averaged.
[c] Average of four brands.
[d] AAS =flame atomic absorption spectrophotometry; NAAS = nonflame AAS.
[e] Median, rather than mean.
[f] References:

(1) Lamm, et al., (1973)
(2) Lamm and Rosen (1974)
(3) Murthy and Rhea (1971)
(4) Mitchell and Aldous (1974)
(5) U.S. Dept. HEW (1975)
(6) Kolbye, et al., (1974)
(7) Ministry of Agriculture, Fisheries and Food (1975)
(8) Brandt and Bentz (1971)
(9) Pinkerton, et al., (1973)
(10) Dutilh and Das.(1971)
11) Schmidt (1974)

The lead content of human breast milk and fresh, homogenized cow's milk (market milk) are quite low in comparison to those milk products which have been canned, evaporated or mixed with additives (i.e., infant formulas). This is as expected, for the lead concentration of the plasma is low and remarkably constant over a wide range of whole blood lead levels (Lamm and Rosen, 1974). Since lead is primarily associated with the erythrocytes rather than the plasma, only a small amount of lead is excreted in milk. Therefore, a naturally low level of lead in milk is expected. Nevertheless, the lead content of milk can be influenced by the lead present in feed and grass and the licking of metal objects or painted surfaces by cows.

Lynch, et al., (1974) also confirmed that very little lead is excreted via the milk, even after administration of substantial amounts of lead. Eleven milligrams of lead, as Pb $(CO_3)_2$, per kilogram of body weight was administered to Holstein cows. This lead level did not produce serious physiological changes despite a large body burden. The lead content of whole milk (5.9 ppb), was lower than the mean lead content (49 ppb) of market milk as determined in a national survey by Murthy, et al., (1967). However, the lead content of milk did not decrease to background levels until 34 days after dosing the cattle.

Pinkerton, et al., (1973) collected human milk from mothers in a breast-feeding club in Cincinnati, Ohio, and bovine milk from individual cows on farms producing milk for the Cincinnati area (Table 8.16). Chemical analyses revealed that human breast milk contained about one-fourth the lead of bovine milk. Median milk lead values were 0.010 and 0.042 ppm, respectively, for humans and cows. Based on this study, a bottle-fed infant could theoretically take in about thirty times the lead of an exclusively breast-fed infant.

Whereas analyses of human milk and homogenized cow's milk have shown low levels of lead, past studies have indicated serious contamination problems in canned evaporated milk. Lead content of other processed milk products (e.g., infant formulas) is generally intermediate between the levels of "natural" milks and those of canned evaporated milk (Table 8.16). Data in 1968 on four brands of evaporated milk indicated concentrations of 0.8 ppm; four brands of infant formulas averaged about 0.4 ppm lead (Murthy and Rhea, 1971). Detailed information on the problem of lead contamination in canned milk products, along with the results of several recent surveys of canned milk products, is given in Section 8.4.5.

8.5.2.5 Beverages, Bakery Products, Sugar and Condiments--

Tables 8.17 to 8.19 present summaries of the lead content of beverages, bakery products, sugar, condiments and miscellaneous foods. With the exception of fruit juices, especially the infant type products, it does not appear that any of these products contain sufficient quantities of lead to be of concern from the standpoint of a total dietary intake. Many fruit juices have a pH in the range of 2.7 to 3.9. Mitchell and Aldous (1974) reported that the mean lead level of canned, infant juice products was 202 micrograms per liter as compared to 35 micrograms per liter in similar bottled products. They explain the higher levels in cans on the basis of the acidity of these products combined with leaching of lead from the seams in cans possessing a high seam to volume ratio.

TABLE 8.17 LEAD CONTENT OF BEVERAGES

Products	Lead Content, ppm	References[a]
Drinking water (U. S.)	0.015[b]	(1)
Lemonade	0.004	(2)
Soda	0.004	(2)
Soft drinks (Holland)	0.02	(3)
Carbonated beverages	<0.008	(4)
Tea	0.20-0.74	(3)(5)
ALCOHOLIC BEVERAGES		
Beer (Cincinnati)	0.01-0.29	(2)
Beer (Holland)	0.03	(3)
Wine (Holland)	0.13	(3)
Brandy (India)	0.05-0.60	(2)
Rum (India)	0.024-0.057	(2)
Dry Gin (India)	0	(2)
Whiskey (India)	0.027	(2)
FRUIT JUICES		
Lime juice	0.30	(4)
Lemon juice	0.57	(4)
Diluted fruit drinks, canned	0.251	(6)
Orange juice, canned frozen concentrate	0.135	(6)
Tomato juice, canned	0.338	(6)
Orange juice, infant	0.308-0.655	(6)
Apple juice, infant	0.207-0.907	(6)

[a] References:
(1) U. S. Environmental Protection Agency (1975)
(2) de Treville (1964)
(3) Reith, et al. (1974)
(4) Meranger (1970)
(5) Ministry of Agriculture, Fisheries, and Food (1975)
(6) U. S. Dept. of Health, Education and Welfare (1975)

[b] Median value.

TABLE 3.18 LEAD CONTENT OF BREAD AND BAKED PRODUCTS[a]

Products	Lead Content ppm[b]
Bread, whole wheat	0.42
Bread, white	0.41
Rolls, hamburger	0.45
Doughnuts, cake	0.45
Biscuit mix	0.24
Flour, all purpose	0.51

[a] Source: Zook et al., (1970).

[b] Mean lead content, dry weight basis.

TABLE 8.19 LEAD CONTENT OF SUGAR, SPICES, CONDIMENTS AND MISCELLANEOUS FOODS

Products	Lead Content ppm	References[a]
Sugar	0.004-0.13	(1)(2)(3)(4)
Corn Syrup	0.21-0.49	(1)
Pepper, black	0.22	(1)(2)
red	0.4	(1)(2)
Ginger	0.08	(1)(2)
Turmeric	0.4	(1)(2)
Mustard seed	0.4	(1)(2)
Cloves	0.15	(1)(2)
Cinnamon	0.03	(1)(2)
Salt	0.2-0.45	(1)(2)
Coffee	0.07	(5)
Cocoa beans	0.15	(5)
Baking powder	5.0-10.0	(1)(2)
Refined soybean oil, canned	0.02-0.10	(6)
Oils, fats, and shortening	0.02	(3)

[a] References:
(1) de Treville (1964)
(2) Haley (1969)
(3) Kolbye, et al., (1974)
(4) Ministry of Agriculture, Fisheries, and Food (1975)
(5) Reith, et al., (1974)
(6) Bergner and Rudt (1968)

8.5.3 Adventitious Lead in Processed Foods

Significant amounts of lead may be introduced in food through processing and packaging. Although the food industry uses stainless steel almost exclusively in processing equipment, substantial amounts of lead are still used in solder for sealing seams in food cans; it was estimated that approximately 4,000 metric tons of lead were used in 1976 (Rathjen, 1978). In 1977, the Assistant Director for Packaging Technology of the Food and Drug Administration (FDA) stated that approximately 13 billion tin plated steel containers used for food have a soldered side seam (Davis, 1977). The solder composition used is 98 percent lead and 2 percent tin (higher tin content solders are now used for evaporated milk). Most of the solder is on the outside of the can and only a small bead is on the interior of the can. Often the can is also lacquered. Nevertheless, absorption of lead still occurs as evidenced by the higher lead levels in canned foods than in similar foods packed in glass or aluminum containers. The Acting Director of the Bureau of Foods, FDA, testified at the public hearing on the proposed national ambient air quality standard for lead that "about two-thirds of the lead in canned food is from the solder" (Roberts, 1978).

A study conducted by the National Canners' Association (now National Food Processors Association) in 1972 was reported by Ewing and Pearson (1974). The study showed that after 1 year of storage in a can, tomato juice averaged 0.36 ppm lead while tomato juices packed in glass averaged 0.26 ppm. Mitchell and Aldous (1974) reported on a study comparing foods packed in metal cans with soldered seams with similar bottled products (Figure 8.10). A comparison of the identical product in different types of containers was possible only in a few cases. Unfortunately, since identical products from the same factory and batch could not usually be obtained, a parallel comparison cannot be presented. The data do, however, clearly indicate a higher mean lead concentration in the canned products (0.167 ppm) than in the bottled products (0.042 ppm), suggestive of the magnitude of the effect of packaging on the lead level of food products.

FDA's Heavy Metals in Foods Survey reported the adult and baby categories in canned foods (see Tables 8.26 and 8.27). Canned tomatoes had the highest mean lead level of all adult foods (0.710 ppm). Such a finding might be anticipated in view of the acidity of tomatos and the rated propensity of acid foods for leaching lead from containers. What is surprising about the data in Tables 8.26 and 8.27 are the high lead levels reported in a variety of other non-acid canned products. Pork and beans, peas, vegetable soup and green beans had lead concentrations of 0.64, 0.43, 0.33, and 0.32, respectively.

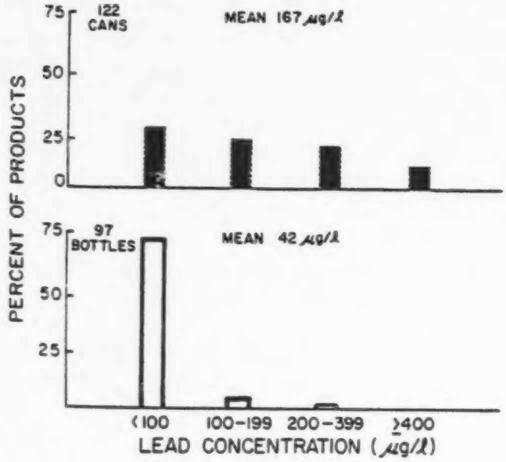


Figure 8.10 Distribution of lead in canned and bottled products. Source: Mitchell and Aldous (1974).

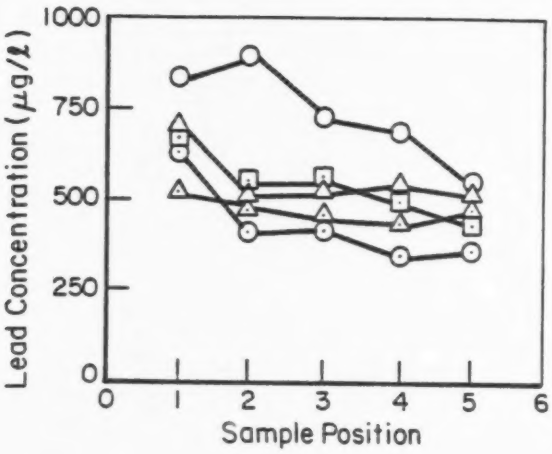


Figure 8.11 Lead distribution in single cans of tomato paste.

Source: Mitchell and Aldous, (1974).
Symbols represent five different cans.

The ability of acid foods to accumulate lead from cans has been the subject of several investigations (Mitchell and Aldous, 1974; National Food Processors Association, 1977). Lead solder was confirmed as a source by a study in which tomato paste (viscous and unmixed) showed decreasing lead concentration as the distance from the soldered seam increased (Mitchell and Aldous, 1974). The results for 5 eight-oz. cans of tomato paste, sampled in 5 positions across a diameter passing through the seam, is shown in Figure 8.11 In all cases, position 1 (nearest the seam) showed a higher level than the opposite side of the can. This effect was particularly evident after the open cans had been stored at room temperature for 24 hours. Here, the lead distribution is even more indicative of leaching from the seam and lead levels up to 5000 μg/l (5.0 ppm) are indicated (Figure 8.12).

The authors hypothesized further, that if lead was entering the can via the seam, there should be a relationship between the seam length/can volume ratio and the lead level of the contents. The data from three groups of cans selected from seam length/volume ratios of 1.25, 0.75-1.25 and <0.75 confirm this suspicion (Figure 8.13). Products in cans having a high seam length to volume ratio (>1.25 and 0.75 to 1.25) had a higher lead content than those in cans having a lower ratio (<0.75). The high ratio cans included mainly baby food and other small juice cans.

A survey conducted in 1974 by the National Food Processors Association (1977) examined lead content of various common vegetables, fruits, and juices prior to canning and after storage in the can for 1-1/2, 3, 6, and 12 months, simulating pre-consumption storage of canned foods. These data (Table 8.20) offer strong support that: (1) food can acquire substantial quantities of lead from cans, and (2) lead concentration in canned food increases with the length of storage. Several foods such as grapefruit juice, orange drink, corn, and tomatos, approximately, doubled their lead content after 1 year in storage.

These data provide some insight into the significant increase in lead content in foods ehich are not particularly agressive towards can seams and interiors. The data in Table 8.20 show that in many cases, most of the lead pickup occurred between the filler bowl of the packing machinery and the initial analysis, which was performed after essentially no storage time, too soon for leaching to have had a measurable effect. The presence of particulate solder in the can would explain this increase; confirmation for this hypothesis is suggested by the fact that extreme variability in lead concentrations were encountered when grab samples were analyzed, and it was found necessary to take the entire contents of a can as a representative sample. Additional support for the hypothesis is provided by the little or no further increase in lead concentrations with storage time observed in some of these kinds of foods. A possible explanation is that this lead results from the fine particulates of solder ("spatter") formed during the soldering and post-soldering brushing of excess solder from the seam.

As recently as 1974, levels of 0.5 ppm were frequently found in canned milk when levels below 0.2 ppm would have been expected on the basis of concentration of whole milk (Kolbye, et al., 1974). These and other available data suggest the need for further research, particularly with regard to solder and

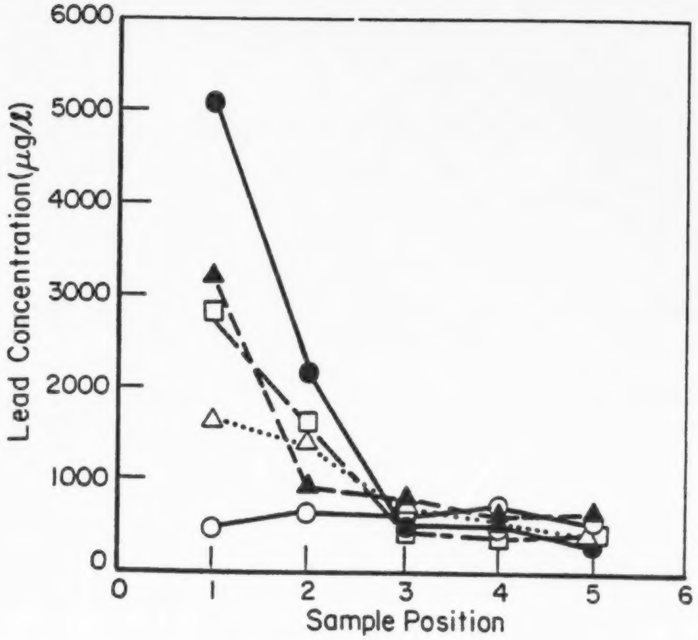


Figure 8.12 Lead distribution in single cans of tomato paste after storage at room temperature.

Second day after purchase, cans kept at room temperature overnight. Symbols represent five different cans.

Source: Mitchell and Aldous (1974).

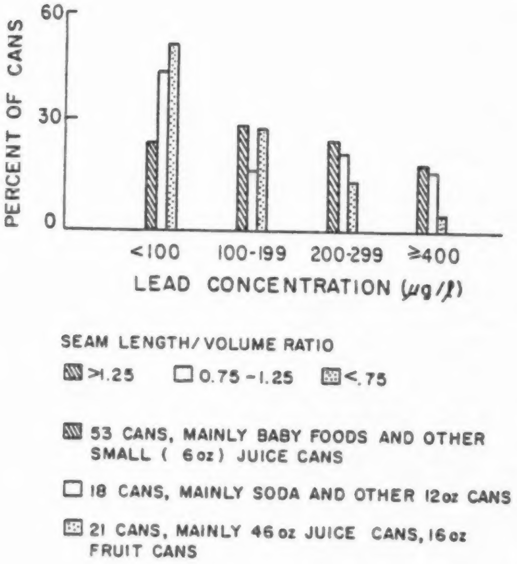


Figure 8.13 Effect of seam length/volume ratio on the lead level of can contents.

Source: Mitchell and Aldous (1974).

TABLE 8.20. VARIATIONS IN LEAD CONCENTRATIONS WITH TIME IN SELECTED CANNED FOOD PRODUCTS [a]

Food Product	Lead Concentration, ppm[b]	Percent Contributed by Can
Corn		
filler bowl	0.05	
initial	0.46	89
6 months	0.59	92
1 year	0.93	95
Green beans		
filler bowl	0.18	
initial	0.48[c]	63
6 months	0.35	49
1 year	0.39	54
Peas		
filler bowl	0.08	
initial	0.33	76
6 months	0.40	80
1 year	0.37	78
Tomatoes		
filler bowl	0.06	
initial	0.31	81
6 months	0.55	89
1 year	0.52[d]	88
Applesauce		
filler bowl	0.08	
initial (mean)	0.27	79
6 months	0.32	75
1 year	0.38	79
Fruit cocktail		
filler bowl	0.08	
initial	0.22[c]	64
6 months	0.21	62
1 year	--	--
Grapefruit juice		
filler bowl		
initial	0.04	100
6 months	0.09	100
1 year	0.30	100
Tomato juice		
filler bowl	0.05	
initial	0.25	80
6 months	0.28	82
1 year	--	--
Orange drink		
filler bowl	0.01	
initial	0.05	80
6 months	0.11	91
1 year	0.11	91

[a] Source: 1974 Lead in Canned Food Survey, National Food Processors Association (1977).

[b] Values are means of approximately 12-50 samples.

[c] Anomalous mean, caused by distribution extremely skewed to the right.

[d] After 9 months.

flux operations in canning evaporated milk. Unlike other food products, evaporated milk traditionally has been packed in a can having a soft solder plug.

Murthy and Rhea (1971) reported that the ratio of tin to lead in the filling solder affected lead uptake by evaporated milk. When the ratio was 60:40 and large amounts of solder were allowed access to the milk, lead uptake of 0.34-1.60 ppm was observed, whereas samples not exposed to solder had 0.05-0.37 ppm. No significant difference was seen in lead uptake by evaporated milk between a 50:50 filling solder and a 10:90 for internal seams. With solder pellets present inside, canned milk showed 0.10-0.54 ppm lead (Engst and Waggon, 1965).

The evaporated milk industry was informed of the need to reduce these levels and the industry has responded by instituting changes in canning operations and improving quality control procedures. A general declining trend from levels as high as 0.5 ppm to 0.1 ppm over the past 5 - 7 years is evident from Table 8.16.

The data obtained by Lamm and Rosen (1974) showed a decrease in lead content of evaporated milk and infant formulas in a 1972-73 survey as compared to that determined in a 1971-72 (Lamm, et al., 1973) survey. These results have quite reasonably been questioned on the basis of a change from flame to nonflame atomic absorption spectrometric analyses (Holliday, et al., 1974; Sarett, 1974). As shown in Table 8.16, Lamm and Rosen's (1974) data shows significantly lower levels for all milk products (including market milk and human milk) than Lamm, et al.'s (1973) study. Thus, much of the discrepancy between the two studies is probably attributable to analytical techniques, rather than an actual decrease of the magnitude suggested (ca 10X). Mitchell and Aldous (1974) obtained higher lead levels in canned evaporation milk (mean, 0.20 ppm; range, 0.01 to 0.28 ppm) using flame atomic absorption spectrometry than did Lamm and Rosen (1974) using a nonflame method (mean, 0.10 ppm; range, 0.04 to 0.22 ppm).

More recent Food and Drug Administration (FDA) analyses indicated that the mean lead content of 80 canned, evaporated milk samples was 0.125 ppm, with a range of 0.02 to 0.37 ppm (Kolbye, et al., 1974). The results of an industrial survey of 3000 canned, evaporated milk samples using a FDA-approved method, showed a mean value of 0.12 ppm Pb, with the highest level in the range of 0.01 to 9.46 (U. S. Department of Health, Education and Welfare, 1975). The food industry has been moving toward elimination of the plug-type metal container. This should aid in minimizing the levels of lead in evaporated milk and related products.

In addition, the importance of evaporated milk in infant nutrition has been in a long-term declining trend over the past 20 years. As reported by Fomon (1974), whereas in 1958 the feeding of about 42 percent of infants through 2 months of age was based on evaporated milk formulas, this had decreased to about 2 percent by 1972. Evaporated milk has largely been replaced by formulas based on soybeans, more nutritionally complete and more easily digested by infants than cow's milk. Evidence in support of this comes from the results of two surveys conducted by one of the major formula producers (Martinez, personal communication, 1977), which indicated the following proportions of infants using evaporated milk formulas:

	Evaporated Milk, percent	
	1976	1971
Hospital period	0.1	0.8
to 2 months	1.0	4.2
3-4 months	2.2	4.0
5-6 months	2.2	3.5

These infant formulas are available either concentrated, requiring dilution before use, or ready-to-use. Both types are packed in regular side-seamed cans, and do not use the plug-type evaporated milk style can.

The use of canned milk (condensed and evaporated) has, in general, also been in a long-term declining trend in the United States, as indicated by U. S. Department of Agriculture 1978 Statistics.

Year	Millions of lb
1967	1,572
1968	1,497
1969	1,376
1970	1,214
1971	1,186
1972	1,102
1973	1,057
1974	1,000
1975	924
1976	862
1977	788

Commercial infant formulas, packed in 8-ounce and 30-ounce seamed cans and in 4-ounce glass nursettes, did not reveal any significant differences in the lead content of the product in either of these types of containers (Lamm and Rosen, 1974). This finding has not been replicated in other studies, however. Lamm and Rosen's (1974) survey, as well as a National Food Processor's Association survey, showed averages of 0.08 and 0.09 ppm, respectively. Similarly, FDA spot analyses showed an average of 0.09 ppm in four major brands of infant formula concentrates. Diluted for use, the lead values would be reduced by 50 percent, making them comparable to market milk.

The consensus of studies conducted by the Can Manufacturer's Institute, in cooperation with the National Food Processors Association (1977), the Food and Drug Administration (U. S. Dept. of Health, Education and Welfare, 1977), and Aldous and Mitchell (1974), suggests that approximately one-half to two-thirds of the lead content in canned foods is attributable to canning, rather than the food itself. In addition, Mitchell and Aldous (1974) have demonstrated that (1) lead solders appear responsible for increasing the lead content in canned foods and (2) substantial quantities of lead may be leached from lead solder seams and incorporated into foods. These results, taken together, indicate the potential for reducing dietary lead through changes in canning technology (e.g. substituting other types of solder, adhesives, or sealing methods for lead solder in can joints or the use of coatings inside the can to act as a barrier to prevent contact between lead soldered seams and food inside the can). Estimates of the effects of instituting these types of changes in reducing both lead intake and (indirectly) body burden of lead among various population groups, are provided in Sections 8.6 and 8.7.

8.6 SOURCE CONTRIBUTIONS TO LEAD INTAKE BY HUMANS

8.6.1 Lead Intake From Air

Inhalation of air is a smaller potential source of lead input for most persons than ingestion of food. Due to the large variation of lead in air with season and locale, the potential intake by this route is extremely variable depending on location and personal physiologic characteristics. Data cited in the NAS report (National Academy of Sciences, 1972) indicate that the median concentration of lead in the air of Los Angeles in 1962 was 2.5 μg/m^3, although some stations in the city recorded individual concentrations as high as 11.4 μg/m^3. In contrast, the concentration of lead in most suburban atmospheres seldom exceeds 0.5 μg/m^3, and for rural areas, 0.1 μg/m^3. As much as a five-fold variation in atmospheric lead exposure has been observed within cities, depending on the location of residence (i.e., downtown Los Angeles (2.5 μg/m^3) or a typical suburb (0.5 μg/m^3). The variability of potential lead intake by inhalation is further increased by the variability in total air inhaled which depends, among other things, on the degree of physical activity and respiratory volume. For example, a man doing light work for 8 hours a day would inhale more than twice (22.8 m^3 vs. 10.8 m^3) as much air as a man at rest all day, as shown in Table 8.21. According to the National Academy of Sciences (1972) calculations, if one superimposes on this range, a 25 fold difference in atmospheric lead concentration between urban and rural air, the potential daily intake of lead could vary from 1.1 μg for a rural man at rest all day to 57.0 μg for a man engaged in light work in downtown Los Angeles, and residing there while not at work. The comparable amount of lead potentially respired by a suburbanite who commutes to the city for light work, is estimated to be 30.6 μg (4.8 + 1.8 + 24.0).

TABLE 8.21 POTENTIAL DAILY INTAKE OF LEAD BY INHALATION[a]

Activity Level[b]	Air Inhaled, m^3	Potential Lead Intake, μg @ 0.1 $\mu g/m^3$	@ 0.5 $\mu g/m^3$	@ 2.5 $\mu g/m^3$
8 Hr. working ("light activity")	9.6	0.96	4.8	24.0
8 Hr. nonoccupational activity	9.6	0.96	4.8	24.0
8 Hr. resting	3.6	0.36	1.8	9.0
Total	22.8	2.28	11.4	57.0

[a] Source: National Academy of Sciences, (1972).

[b] Of a "standard man" weighing 70 kg, 20-30 years old, 175 cm tall, and having a surface area of 1.8 m^2.

Interestingly, as in the case of dietary intake, potential respiratory intake in infants is greater than that of adults on a μg/kg body weight basis for exposure at comparable concentrations of airborne lead. The basis for this is the greater volume of air respired by children per unit of body weight. A 1-year-old child inhales 6 m^3 of air per day compared with the 23 m^3 per day inhaled by an adult (National Academy of Science, 1972). The significance of this proportionally greater inhalation of lead cannot be properly evaluated as it is presently unknown whether the higher potential intake may be accompanied by a correspondingly higher, equal, or lower rate of excretion than adults.

Although it is generally agreed that for most individuals, dietary lead presents a more important (quantitative) source of exposure than air, there are many uncertainties concerning their relative contributions to body burden. One of the chief reasons for these uncertainties is the lack of specific knowledge concerning the fate of inhaled lead once it enters the lungs, and the factors which may modify absorption rates. The contributions of food and water to daily lead assimilation, in contrast, have been studied extensively in detailed, long-term balance studies such as those of Kehoe (1961). The few studies which have examined inhalation of lead in humans are reported below.

Mehani (1966) conducted an investigation to determine the degree of initial deposition and retention of inhaled lead in the lungs. That study reported an average deposition of 37 percent for non-industrially exposed men and 39 and 47 percent for two groups of industrially exposed men, respectively. The particle size distribution was not reported.

In general, the information concerning the size distribution of lead particles in the environment indicates that particle sizes of mass median equivalent diameters (MMED) of 0.1 - 1.0 μm should be of the greatest concern; the average MMED in the general atmospheric environment is about 0.25 μm (Robinson and Ludwig, 1967).

Kehoe (1961) presented the results of a study in which both the deposition rates in the lungs and the particle size was reported. Lead sesquioxide produced by burning tetraethyllead was the source of the exposure; the particles generated were classified according to two size groups: an average diameter of 0.05 μm with 90 percent from 0.02 - 0.09 μm; and a second group having a median diameter of 0.9 μm, with 90 percent less than 2 μm in size. The 0.05 μm size corresponds to an MMED of 0.25 μm reported for the general atmospheric environment by Robinson and Ludwig (1967). The concentration of lead used by Kehoe (1961) was 150 $\mu g/m^3$, much higher than that encountered in the general environment, but not atypical of industrial exposure levels. The deposition of the smaller particles in the respiratory tract was 36 percent [which closely approximates the 37 percent estimated by Mehani (1966)] while that of the larger particles (MMED = ∿ 2.9 μm was 46 percent.

Nozaki (1966) utilized a variety of particle sizes ranging from 0.05-1 μm. Rates of deposition under both rapid, shallow, and slow, deep, respiration were determined. For particles having a diameter of 0.05 μm, the total deposition in the respiratory tract under conditions of slow deep breathing was 42.5 percent but, as shown in Table 8.22, all deposition rates were appreciably lower during rapid, shallow conditions of respiration.

TABLE 8.22 DEPOSITION OF LEAD INHALED BY MAN[a]

10 Respirations/min; 1,350 cc Tidal Air		30 Respirations/min; 450 cc Tidal Air	
Particle Diameter, μm	Particle Deposition, percent	Particle Diameter, μm	Particle Deposition; percent
1.0	63.2	1.0	35.5
0.6	59.0	0.6	33.5
0.4	50.9	0.4	33.0
0.2	48.1	0.2	29.9
0.1	39.3	0.1	27.9
0.08	40.0	0.08	26.5
0.05	42.5	0.05	21.0

[a] Source: Adapted from Nozaki (1966) by National Academy of Sciences (1972).

In summary, the bulk of available experimental evidence suggests that the deposition in the respiratory tract of lead from the ambient air is approximately 35-40 percent (National Academy of Sciences, 1972). Again, it must be noted that these results may not be generalizable to groups such as women and children not included as experimental subjects in these studies. The NAS panel concluded, "knowledge concerning the contribution of respired air to the total amount of lead entering the body is insufficient to allow anything better than general approximations. A reasonably sound estimate can be provided for the fraction of the inhaled lead which is deposited in the airways, but very little is known concerning its fate once deposited".

Under certain conditions, inhaled lead of large particle size may contribute to gastrointestinal absorption. Lead particles deposited in the nasorpharyngeal region may be swallowed or ejected by nose blowing or expectoration. Similarly, ciliarymucus transport may cause migration upward to the pharynx where swallowing or expectoration may occur, or they may enter the systemic circulation. Particles deposited in the alveolar bed may be phagocytized by migratory macrophages and conveyed back up the airways to be swallowed and passed through the gastrointestinal tract, according to the National Academy of Sciences' report (1972). A single study of Kehoe (1961), attempted to quantify the importance of this mechanism under experimental conditions. Kehoe determined the excretion of lead in urine before, during and after termination of inhalation of small and larger particles of lead oxide (average MMED 0.25 μm; median MMED 2.9 μm). During long periods of lead inhalation, Kehoe found that excretion of lead of both size groups increased. Only during inhalation of the larger particles was there an increase in fecal lead excretion. This increase was presumably attributable to retrograde movement of larger particles from the lungs to the nasopharynx followed by swallowing. National Academy of Sciences (1972) estimated that 40 percent of the lead deposited in the airways was transferred to the gastrointestinal tract; this transfer probably involved the "large" particles (in the 2.9 μm MMED range). The contribution of "respiratory" lead intake to gastrointestinal absorption under normal environmental conditions is unknown.

Various estimates of absorbed lead dosage from air have been derived. Given 37 percent deposition and total retention/absorption, inhalation of 23 m^3 of air per day, and a lead concentration of 2.5 $\mu g/m^3$, 21 μg of lead per day originating in the air would be absorbed compared with, roughly, 30 μg/day originating in foods and water (National Academy of Sciences, 1972). At the maximum concentration permitted by the new lead air quality standard (1.5 $\mu g/m^3$), daily absorption of inhaled lead would be reduced from 21 μg to 12.6 μg, and at the 1975 geometric mean national urban air lead concentration of 0.75 $\mu g/m^3$ (Table 8.3) daily lead absorbed from air would be further reduced to 6.3 μg.

Other investigators, as cited by Ewing and Pearson (1974), suggest that the previous assumption of 23 m^3 of air inhaled per day is too high and estimates would be more appropriately based on lower volumes of respired air. Accordingly, Ewing and Pearson (1974) estimated that a lead dosage of 20-25 μg would result from an urban adult breathing air containing 3 μg/m^3 and respiring about 15 m^3 of air per day. This estimate would be similarly reduced at the lower lead concentrations, more representative of current urban air lead concentrations.

Data on percent absorption by children are scant, due, in part, to the difficulties in the participation of child subjects in experimental studies. For the purposes of estimating relative contributions from the various media, an average absorption of 40 percent for inhaled lead was adopted for both children and adults.

8.6.2 Lead Intake From Drinking Water

The environmental distribution and concentrations of lead found in U. S. waters are discussed in Section 8.3.2. In this section, the relationship of these lead levels to the total human exposure profile is considered. As noted in Section 8.3.2, very few water supplies have lead concentrations in excess of the 50 μg/l drinking water standard. The median lead concentration in municipal water supplies appears to be in the range of 10 μg/l, based on the analytical data described in Section 8.3.2. A few localities in the U. S., notably metropolitan Boston, have higher exposures due to very soft water and the presence of lead in domestic piping systems.

With the exception of a very few localities, (e.g., Boston), it is apparent that drinking water contributes a minor percentage of ingested lead for the average adult. Based on an examination of data from nationwide water quality surveys, the National Academy of Sciences (1972) concluded that it is doubtful that many people drink water containing greater than 50 μg of lead/liter with any consistency.

The absorption characteristics of ingested lead compounds have been studied by many investigators, beginning with the classic work of Kehoe in the early 1960's. For adults, most of the estimates appear to cluster in the neighborhood of 10 percent, and this value was adopted for both food and water for the estimation of relative contributions of the several media.

Assuming, on the average, a lead content in drinking water of 10 to 20 μg/l and a daily consumption of 1 to 2 liters, the lead intake would range between 10 and 40 μg/day, of which about 10 percent, or 1 to 4 μg, would be absorbed. This intake would represent less than 10 percent of the World Health Organization (W.H.O.) recommended acceptable total intake of 3 mg/wk (429 μg/day) for adults. Depending on the levels of intake from other sources, water may contribute between 10 and 20 percent of the daily absorbed dose of lead for the average U. S. adult.

The efficiency of absorption of ingested lead by children is much greater than the 10 percent of adults. Alexander (1973;1974) showed a value of 53 percent. In a recent broader study conducted by Ziegler, et al., (1978), the average absorption for 61 balance studies on children ranging from 14 days to about 2 years in age was 41.5 percent. This latter value was adopted for children for both ingested food and water.

8.6.3 Lead Intake From Soil and Other Non-Food Items

It is generally accepted that ingested dietary lead constitutes the body's largest direct source of lead. Lead intake from soil and dust is a negligible problem for adults and can be neglected. However, this may constitute a very important route for lead intake by infants and pre-school children, as suggested in Section 8.4.3, especially for young children with pica habits or prone to hand-to-mouth activity. Lead intake may be estimated based on lead concentrations in dirt and dusts and the mouthing behavior of the child. Unfortunately, there is a great deal of uncertainty on the availability of the contained lead and, hence, of its absorption. It is, undoubtedly, less well absorbed than lead in ingested food or water; for purposes of calculation, a conservative assumption might be that it is 3/4 as well absorbed, i.e., about 30 percent absorption.

In the study by Day, et al., (see Section 8.4.3), it was found that the average concentration of lead in urban dust was 960 ppm (860 ppm median). The authors found, through direct measurements, that from 5 to 50 mg of dirt can be transferred from a child's hands (dirty from 30 minutes of normal play) to a "sticky sweet". Therefore, approximately 100 μg of lead could be ingested via consumption of two sweets.

Day, et al., calculated that children aged one to nine ingest, approximately, 165 μg of lead per day in food and water. Consumption of two sweets during normal play would add to this amount considerably.

In the similar investigation by Lepow, et al., (1975) (see Section 8.4.3), the transfer of lead from children's hands to their mouth was also observed and measured. Hand dirt had an average lead concentration of 2400 ppm. Most of the children were observed to have their hands or non-food objects in their mouths frequently. A reasonable estimate of mouthing frequency was established at ten times per day. The authors estimated, using the above figures, that a child exhibiting this mouthing behavior and playing in a similarly contaminated environment could ingest 240 μg of lead per day, thus approaching the maximum permissible daily intake from this source alone.

Given the combination of the high lead content of the house dust and the mouthing proclivities of the study children the high blood leads of these children are, perhaps, not surprising, and serve to point up the fact that paint pica is not the only danger for small children living in substandard housing. It is fortunate that the large majority of children are not so exposed, else high blood lead levels would be far more prevalent.

A non-food item which may be a contributor to lead intake by adults is lead from cigarette smoke. As pointed out by Mahaffey (1978), the concentrations of lead in tobacco can vary widely; Cogbill and Hobbs (1957) found concentrations up to 39 ppm. Investigation of lead to arsenic ratios implicated lead arsenate as the probable source of the lead in cigarettes. Surprisingly, little information on the lead content of tobacco smoke is available. Cogbill and Hobbs estimated that the amount of lead transferred to mainstream smoke was about 4-6 percent of the lead concentration in the cigarette, corresponding to about 1-2 μg per cigarette. Patterson (1965) estimated about 0.8 μg of lead is contained in each cigarette, resulting in about 6.4 μg of lead absorbed per day for a pack-a-day smoker. The 40 percent retention rate assumed by Patterson does not appear to be documented specifically in the literature; selection of this figure is probably based on analogy to deposition/retention rates of lead observed in experimental inhalation studies (National Academy of Sciences, 1972).

A point to note here is that the references cited on lead content of tobacco in cigarettes are based on 15 to 20-year-old data, from a period when lead arsenate was a widely used insecticide. As described in Section 4.3.4.2.3, in recent years lead arsenate has been supplanted by organic pesticides to the extent that it is no longer manufactured in the U. S. Thus, there appears to be some question whether cigarettes may still be a significant source of lead intake for smokers. There do not appear to be any recent studies bearing on this question. For purposes of calculation, the 40 percent absorption for inhaled lead is assumed to be also applicable to lead from cigarette smoke, although there does not appear to be much experimental data on this either.

8.6.4 Lead Intake from Foods and Beverages

Daily dietary lead intake is determined by the amount and kinds of food and beverages ingested and by the lead content of each of the various components of the diet. As discussed earlier, a portion of inhaled lead may also contribute to ingested lead if the inhaled lead moves from the lungs to the nasopharyngeal area where it may be swallowed.

Several methodologic approaches have been used to derive estimates of dietary lead intake. These include: (1) analysis of "typical diet" replicates, (2) institutional total diet studies (i.e., composites), and (3) balance studies, which measure both intake and excretion of individuals maintained under experimental conditions, and (4) estimates based on measurement of fecal lead excretion.

Total dietary intake estimates, utilizing the techniques above, place the typical adult daily lead intake in the range of 90-500 μg/day with most of the estimates converging on the 150-300 range. Much of the variation observed in these studies is due to difference in absolute food intake. For example, in a nationwide study of institutionalized children, Murthy, et al., (1971) reported that the children's average food consumption ranged from 1.18 kg/day to 2.55 kg/day depending on the institution. Similarly, within single institutions, individuals ranged between 0.92 - 3.10 kg/day of food. This threefold range of intake level (or perhaps more) presumably applies to the general population as well.

Kehoe (1961), Schroeder, et al., (1961), Harley (1970), Lewis (1966), as cited in the National Academy of Sciences (1972) have published estimates ranging from 100-500 μg/day based on food intake levels of about 2,000 g of food and beverages per day. Estimates of lead intake conducted over the past 30 years have been remarkably similar, suggesting that any trends in lead consumption, if present, must be extremely subtle (National Academy of Sciences, 1972; Ewing and Pearson, 1974).

Harley's (1970) study is particularly interesting. Lead concentrations in various foods in New York City were determined. These values were then multiplied by U. S. Department of Agriculture estimates of food consumption. Findings of the study revealed a weighted lead intake of approximately 280 µg/day; a food consumption of 1.75 kg/day per person; and a mean lead concentration in food of 0.163 ppm.

Composition of the diet determined by food choice also influences lead intake. Lewis (1966) as cited by the National Academy of Sciences (1972), points out that no food or group of foods is either a large or a constant contributor to lead in man, because man's diet is composed of a wide variety of individual items, and various food contribute various amounts of lead. Lewis (1966) estimated the average daily dietary intake of lead at about 300 µg, with a range for most people of 100-2,000 µg. The range can vary markedly from person to person and from city to city, on the basis of choice, opportunity, and specific habits.

The Food and Drug Administration (FDA) conducts two major food surveillance programs in the U. S. FDA's Total Diet Studies is a generalized program to determine the levels of pesticide residues, PCB's, heavy metals (including lead), and other contaminants in the diets of U. S. consumers. The information generated by this program is based on continual monitoring of total diet samples or "market baskets" from retail markets in 30 locations nationwide (U. S. Dept. of Health, Education and Welfare, 1977). The specific foods collected are designed to simulate the 2 week diet of a 15-20 year old male, statistically the largest food consumer. Each market basket consists of 117 foods representative of 12 food groups (i.e., dairy; meat, fish and poultry, fruits, etc.). The composition of the market baskets vary somewhat both in relative amounts of various commodities and also there is minor variation in the commodities sampled from region to region. These variations are intended to reflect regional differences in diet.

Foods sampled under the Total Diet Studies program are purchased from retail markets in amounts and types specified by FDA. The foods are then prepared (washing, peeling, cooking, baking, etc.) according to uniform instructions. Foods are then divided into the 12 food groups and composited for sampling by combining the foods in proportions reflective of average consumption levels for each commodity as determined by a U. S. Department of Agriculture household food consumption survey. (U. S. Department of Agriculture, 1965). The lead content of each of the 12 food groups composites is then measured. Consequently, the program does not provide data on the lead content of individual foods.

Total Diet Studies, or "Market Basket Studies" have shown a very generalized distribution of lead in foods, with no single food group or source assuming overwhelming importance. In general, however, highest levels are seen in vegetables, particularly leafy ones (Mahaffey, et al., 1975; U. S. Department of of Health, Education and Welfare, 1977). Market Basket Studies have consistently revealed the highest quantities of lead in food classes representing vegetables (either leafy, legume, or root) and in fruits and garden fruits as shown in Tables 8.23 and 8.24. The fruits and garden fruits together compose only 10.4 percent of the total diet. However, they provide 34 percent of daily lead intake. Similarly, the vegetable groups (i.e., legume, root or leafy vegetables) account for only 5.7 percent of total daily food intake, yet they contribute 44 percent of daily intake. Together, fruits and vegetables (Food Classes V, VI, VII, VIII, and IX) account for almost 80 percent of total daily food intake. Estimated dietary intake from all food groups is shown in Table 8.25.

TABLE 8.23 ESTIMATED DIETARY INTAKE OF LEAD BY FOOD CLASS[a]

		Lead	
		µg/day	Percent total
I	Dairy products	0.0	0.0
II	Meat, fish, and poultry	4.00	6.6
III	Grain and cereal	4.16	6.9
IV	Potatoes	0.70	1.2
V	Leafy vegetables	3.03	5.0
VI	Legume vegetables	18.80	31.1
VII	Root vegetables	3.83	6.4
VIII	Garden fruits	11.36	18.8
IX	Fruits	9.49	15.7
X	Oil and fats	0.67	1.1
XI	Sugars adjuncts	0.55	0.9
XII	Beverages	3.81	6.3
	Totals	60.4	

[a] Based on Fy 1973 Total Diet Survey data of the Food and Drug Administration as reported by Mahaffey, et al., (1975).

TABLE 8.24 RANK ORDER OF LEAD FINDINGS BY FOOD CLASS; FY 1973[a,b]

Food Class	Percent Positive	Rank Based on Mean Highest Concentration	Mean Concentration ppm[c,d]
Legume vegetables	100	1	0.26
Garden fruits	87	2	0.12
Root vegetables	83	3	0.11
Grains and cereal products	73	4	0.10
Leafy vegetables	83	5	0.05
Fruits	73	6	0.042
Meats, fish, and poultry	77	7.5	0.013
Oil, fats, and shortenings	50	7.5	0.013
Sugars and adjuncts	63	9	0.0067
Potatoes	57	10.5	0.0033
Beverages	17	10.5	0.0033
Dairy products	33	12	(0)[e]

[a] Source: Mahaffey, et al. (1975). (FDA Total Diet Study)

[b] Sampling locations distributed over the United States

[c] Lead determinations made by flameless atomic absorption spectro photometry

[d] Based on 30 "Market Basket" samples of each commodity

[e] Traces count as positive occurrences but with no ppm value.

Of the various methods of estimating daily dietary lead intake, the FDA Total Diet studies have consistently provided the lowest estimates. For example, the FY 1973 figure for lead intake was 60.4 μg/day. The comparable figure for FY 1974 was 90.2 μg/day. Changes in the method of reporting which allowed a lower minimum level to be reported in 1974, rather than actual increases in intake, are thought to account for the 30 μg/day discrepancy between FY 1973 and FY 1974 figures (U. S. Department of Health, Education and Welfare, 1977). Kolbye, et al., (1974) has severely criticized the accuracy of these estimations, suggesting that analysis of the foods as composites rather than separate foods leads to substantial under-estimation of the quantities present, because trace values in the composites (near the detection limits) have been counted as zeros. Determination of lead in foods, in general, has presented difficulties because (1) the limits of detection for lead are high relative to the other heavy metals (0.1 ppm for lead vs. 0.01 ppm for cadmium and mercury), (2) the quantities typically present in foods are low in relation to detection limits.

Under differing assumptions about the actual values of these trace concentrations, Kolbye, et al., 1974, showed that a four-fold difference could exist in the estimates of intake based on the 1973 Total Diet Study data (Table 8.25).

TABLE 8.25 THE EFFECTS OF VARIOUS ASSUMPTIONS OF THE VALUE OF TRACE AND NON-DETECTIONS ON ESTIMATES OF DIETARY INTAKE[a]

Study Title and Method of Calculation	Calculated μg Lead Per Day for Teenage Male
FY 73 Total Diet Studies (Traces and Non-detections = 0) (See Table 8.11)	60
FY 73 Total Diet Studies as Reported by Kolbye, et al., (1974)	
(with traces assumed = 0.09 ppm and non-detections assumed = 0.00 ppm)	159
(with traces assumed = 0.09 ppm and non-detections assumed = 0.05 ppm)	233
FY 74 Total Diet Studies[b]	
(with traces and non-detections = 0	90
(with traces assumed = 0.02 ppm, which was half of lowest quantitated levels in FY 74 and non-detections = 0	95
FY 74 Heavy Metals in Foods Survey	
(using median levels of surveyed foods)	141
(using mean levels of surveyed foods)	254

[a] Source: U. S. Department of Health, Education and Welfare, 1977.

[b] The analytical method was the same during FY 73 and FY 74. However, in FY 74, after additional experience in use of the method, the laboratory was requested to estimate detected values lower than the 0.1 ppm. In FY 73, 0.1 ppm was the lowest reportable level.

The range of values derived was 60-233 μg lead per day for FY 1973. A similar analysis of FY 1974 data revealed a range of 90-254 μg/day depending on the treatment of "trace" findings and the choice of median or mean levels for calculations (U. S. Department of Health, Education, and Welfare, 1977). Thus, analytical sensitivity of the method becomes a factor along with how "traces" or "non-detections" are treated. These factors significantly alter the calculation of daily intake levels.

In contrast, estimates of dietary lead intake derived from replicate diets and fecal lead excretion in strictly controlled metabolic studies, place lead intake from approximately 150-300 μg Pb/day depending on total food intake (Kehoe, 1961; Schroeder and Tipton, 1963).

In view of the difficulties in evaluating food composites, in 1974 FDA instituted a second food monitoring program called the Heavy Metals in Foods Survey, to examine lead in 41 different staple items of infant, child, and adult diets. The last entry in Table 8.25, 141 - 254 μg/day is based on this survey. The Heavy Metals in Foods Survey is somewhat similar to the Total Diet Studies program except that fewer food commodities are sampled and the food products are analyzed individually. The Heavy Metals in Foods Survey also distinguished between canned and non-canned foods in each commodity. A total of 41 foods are surveyed in this program. The specific commodities sampled are selected on the basis of: (1) relative importance to the diet. (2) past indications of high lead levels in the food, (3) balanced coverage of raw and processed foods. The 41 foods surveyed are thought to account for at least 71 percent of the daily diet. Hopefully, this survey will provide important information on the overall lead levels in infant and adult diets and identify areas in which technological improvements need to be made and/or guidelines or regulatory action may be appropriate.

Lead content of the 41 adult and baby foods sampled are reported in Tables 8.26 and 8.27. Although only one year's data from the Heavy Metals in Foods Survey has been released (FY 1974), several significant findings have emerged with respect to lead in foods.

The mean lead content of canned commodities (averaged over all canned foods surveyed) is between two and three times the lead content of all the non-canned products. The respective average lead content of the canned and non-canned groups were 0.376 ppm and 0.156 ppm (U. S. Dept. of Health, Education, and Welfare, 1977).

For both adults and children, approximately 30 percent of daily lead intake comes from canned foods although they represent only about 11 percent of the diet by weight. These two findings underscore the importance of canned foods as a potentially controllable source of lead intake. Further discussion of this topic is included in Seciton 8.6.7.

TABLE 8.26 LEAD CONTENT OF ADULT FOODS SAMPLED BY FDA HEAVY METALS IN FOODS SURVEY, FY 1974[a,b]

		Concentration, ppm		
	No. of Samples	Mean	Range	Upper Tolerance Limit
Carrots, fresh	70	0.205	0-3.54	1.234
Lettuce, raw	69	0.130	0-2.02	0.712
Potatoes, raw white	71	0.050	0-0.762	0.251
Butter	71	0.068	0-0.151	0.455
Margarine	71	0.068	0-0.37	0.159
Eggs, whole fresh	69	0.174	0-4.02	0.792
Chicken, fryer, raw whole or raw cut-up	71	0.127	0-1.30	0.507
Bacon, cured raw sliced	71	0.099	0-1.685	0.598
Frankfurters	69	0.198	0-2.87	1.087
Raw liver, beef	71	0.089	0-1.03	0.387
Hamburger, raw ground beef	71	0.248	0-8.29	1.396
Roast, chuck beef	71	0.071	0-0.54	0.309
Wheat flour, white	70	0.052	0-1.62	0.400
Cornmeal, yellow	70	0.143	0-2.54	0.563
Rice, white milled	71	0.104	0-2.76	0.662
Breakfast cereal ready-to-eat	68	0.107	0-2.18	0.526
Sugar, refined beet or cane	71	0.031	0-0.44	0.146
Bread, white	70	0.084	0-0.91	0.347
Beets, canned	71	0.381	0-3.47	1.448
Tomato juice canned	71	0.338	0-3.08	0.803
Mixed vegetable juice, canned	70	0.215	0-1.76	0.770
Soup, vegetarian vetetable	71	0.331	0-2.85	1.382
Orange juice, canned frozen concentrate	71	0.135	0-5.2	1.351
Green beans, canned	71	0.318	0-1.34	0.860
Beans canned with pork and tomato	71	0.638	0-4.74	1.959
Peas, canned	70	0.425	0-6.95	1.912
Tomatoes, canned	71	0.710	0-4.81	2.375
Diluted fruit drinks, canned	71	0.251	0-3.18	1.137
Peaches, canned	71	0.417	0-2.908	1.097
Pineapple, canned	71	0.402	0-7.10	1.306
Applesauce, canned	71	0.320	0-3.03	0.898

[a] Source: U.S. Department of Health, Education and Welfare (1975).

[b] Sampling locations distributed throughout the United States.

[c] Upper limit of 95% confidence interval; 95% of individual values in the population samples are at or below this limit.

TABLE 8.27 LEAD CONTENT OF BABY FOODS SAMPLED BY FDA HEAVY METALS IN FOODS SURVEY, FY 1974[a,b]

Food Product	No. of Samples	Concentration, ppm		
		Mean	Range	Upper Tolerance Limit
Vegetables and Beef	71	0.070	0-0.67	0.245
Mixed Vegetables	71	0.072	0-1.12	0.406
Spinach	69	0.085	0-0.55	0.270
Orange Juice	71	0.375	0-3.66	1.533
Apple Juice	71	0.321	0-1.47	0.885
Applesauce	71	0.160	0-1.37	0.547
Pears	71	0.079	0-0.494	0.212
Peaches	71	0.094	0-0.458	0.259
Apricots	71	0.082	0-0.63	0.283

[a] Source: U. S. Department of Health, Education and Welfare, (1975).

[b] Sampling locations distributed throughout the United States.

[c] Upper limit of 95 percent confidence interval; 95 percent of individual values in the population samples are at or below this limit.

8.6.5 Relative Contribution of Various Media to Body Burden

8.6.5.1 Adults--

Of the major pathways of exposure to lead (air, food, water and sometimes tobacco smoke), food represents the major contributor to body burden for the general population. As indicated, general population as defined here refers to normal, non-occupationally exposed adults and to older children whose exposure through dust, dirt, and paint is negligible. Individuals exposed to unusually high ambient concentrations of lead (say above 2.5 $\mu g/m^3$ in air) such as those living in close proximity to major lead emitting industries or extremely high density traffic are possible exceptions. For those groups, air may constitute an equal or, perhaps, more important source of exposure. It should be noted that unusual conditions of exposure outlined above apply to a very small percentage of the population, consequently, the bulk of the present discussion is directed toward the more typical lead exposure profile. A discussion of the special problems of high risk groups is deferred until Section 9.

Although food is the principal contributor to absorbed lead, the proportionate contributions of various media (air, food, cigarettes, water) is quite variable depending on the level of exposure from ambient air. Figure 8.14 shows that food contributes at least one-half of the body retention or uptake for the urban man exposed to 1.3 $\mu g/m^3$ in air. For the nonsmoking rural inhabitant (ambient air at 0.05 $\mu g/m^3$) food contributes as much as 90 percent of

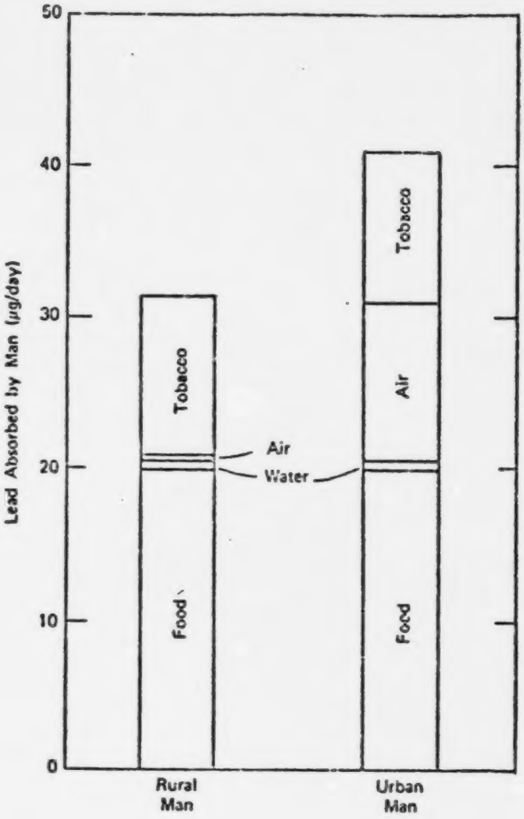


Figure 8.14. Lead absorbed by man.
Source: Ewing and Pearson (1974)
Adapted from Patterson (1965).

absorbed lead dose. Note also that the absolute quantity of lead absorbed is approximately 10 µg per day greater for the urban resident as opposed to the rural inhabitant.

Table 8.28 compares the amount of lead absorbed (µg/day) from air, water, food and cigarettes under various levels of daily intake for young adult males for each of the media. Assumptions regarding lead concentrations in the various media and the fraction of intake actually absorbed are based on data presented in previous sections regarding these parameters and especially on information derived from the comprehensive reviews of Patterson (1965), National Academy of Sciences (1972) and U. S. Environmental Protection Agency (1977).

TABLE 8.28 THE RELATIVE CONTRIBUTIONS OF VARIOUS MEDIA TO LEAD INTAKE AND ABSORPTION: ADULT MALES

Media	Daily Intake	Lead Concentration	Daily Lead Intake, μg/day	Percent Absorbed[a]	Daily Lead Uptake Absorption, μg/day
Food	Standard Diet in USDA consumption data as used in Market Basket Studies, kg/day	Varies with diet composition. Average lead content approximately 0.1 μg/g	150	7.5	11.25
			200		15.00
			250		18.75
			300 (254)		22.50
			400		30.00
Water	1.0 ℓ	20 μg/ℓ	20	10	2.0
	1.5 ℓ		30		3.0
	2.0 ℓ		40		4.0
Air	20 m^3	0.10 μg/m^3	2.0		0.8
		0.25	5.0	40	2.0
		0.50	10.0		4.0
		1.00	20.0		8.0
		1.50	30.0		12.0
		2.00	40.0		16.0
		2.50	50.0		20.0
		3.00	60.0		24.0
		3.50	70.0		28.0
		4.00	80.0		32.0
		4.50	90.0		36.0
		5.00	100.0		40.0
Tobacco Smoke	10 cigarettes	0.8 μg/cigarette	8	40	3.2
	20 cigarettes	0.8	16		6.4
	30 cigarettes	0.8	24		9.6

[a] Source: Battelle estimates derived from literature

[b] FDA estimate for lead intake of teenage male (See Table 8.25).

The companion table (Table 8.29 shows the total absorbed dose of lead received per day under a variety of ambient air conditions). Note that non-air sources including food and water (and cigarettes, for smokers) contribute 22.0 and 28.4 μg/day, respectively, to abosrbed lead. For both smokers and nonsmokers, the proportionate contribution from ambient air increases as ambient air lead increases. At approximately, 2.75 μg/m^3 ambient air supplies as much absorbed lead per day as does diet. For smokers, the equivalent figure is about 3.5 μg/m^3 lead in air. The absolute quantity of absorbed lead nearly triples from its baseline value (at 0.01 μg/m^3 in air) to 62.0 μg/day at ambient air lead levels of 5.0 μg/m^3 for nonsmokers. For smokers, the increase is less dramatic, however, daily absorbed lead more than doubles, increasing from its baseline value of 28.4 μg/day to 68.4 μg/day absorbed when ambient lead in air is at 5.0 μg/m^3. The range of 0.1 to 4.0 μg/m^3 of lead in air approximates the usual range of ambient conditions for general populations. As indicated in Section 8.2.1, only 5 stations reported mean annual lead concentrations of over 2 μg/m^3 and none of the stations reported over 3.0 μg/m^3.

Figure 8.15 portrays the data from Table 8.28, assuming an absorbed dose from food of 254 μg/day, 3.0 μg/day from water and 6.4 μg/day from cigarettes. The area beneath each of the lines (i.e., Food, Air, Smoke, and Water) corresponds to the percent contribution to absorbed lead dose for each media for a given ambient air concentration of lead. The total of all media for any given air lead concentration is 100 percent. The actual absorbed dosage in μg/day for each ambient air concentration may be derived from Table 8.28. As indicated in Figure 8.15, at low to moderate levels of lead in air, food and water account for a very large proportion of absorbed lead dose for the general adult population. As air lead levels increase, however, air takes on an increasingly prominent position and actually surpasses absorbed lead from food and water at approximately 2.75 μg/m^3. Beyond that point (>2.75 μg/m^3 in air), food diminishes in importance whereas air increases. 2.75 μg/m^3 is well above average annual means currently measured even in most densely urbanized areas; lead levels in the vicinity of point sources such as primary and secondary smelters may exceed 2.75 μg/m^3, however. Except at the lowest ambient air lead concentrations, water and cigarettes maintain a low and rather uniform contribution to absorbed dose. If lead arsenate is the cause of the dose from cigarettes, this source may be overestimated.

8.6.5.2 Children--

As suggested in previous sections, the exposure patterns of children differ dramatically from those of adults. Several studies have demonstrated that gastrointestinal absorption of lead is much more efficient in young children than in adults. Ziegler, et al., (1978) found a mean absorption rate of 41.5 percent based on metabolic balance studies of children. This implies that at a given lead concentration in food, a child will actually absorb more than 4 times more lead than an adult consuming the same item. Also, the composition of children's diets is different from adults (see Table 8.33); children consume larger amounts of food than adults on a grams/kg body weight/day basis. Inputs from smoking can be ignored for children. Perhaps, the most important difference in exposure patterns between children and adults is that dust and dirt constitute a significant route of exposure for children.

Hand-to-mouth activity (e.g., thumb sucking and finger licking) is a normal behavioral characteristic of children through five years of age. This can

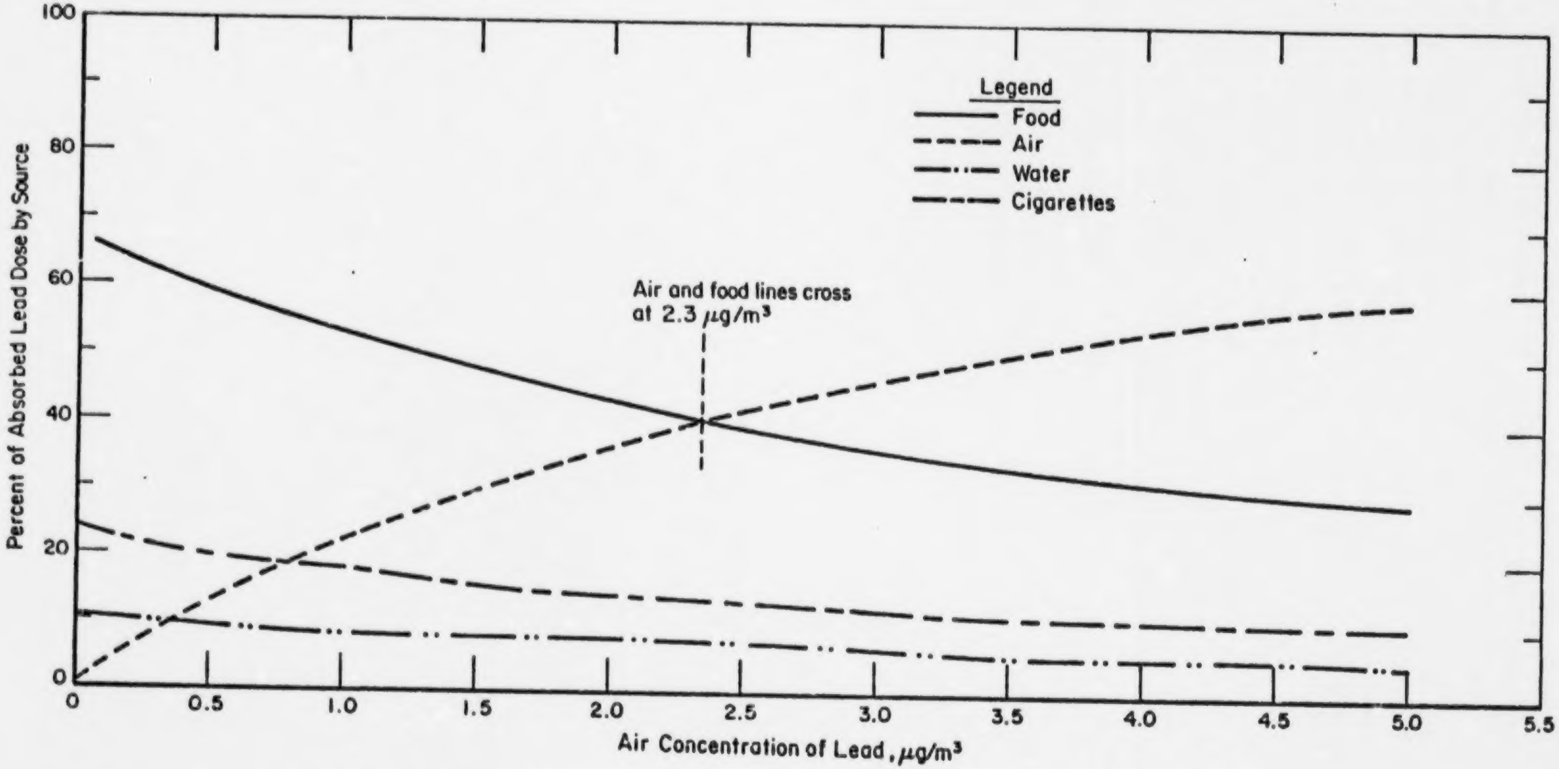


Figure 8.15 The relative contribution of various media to total absorbed lead dose.

Source: Battelle-Columbus estimate, based on 254 μg/day from food, and 30 μg/day from water.

TABLE 8.29 ESTIMATED TOTAL ABSORBED DOSE AT VARYING AIR LEAD CONCENTRATIONS[a]: YOUNG ADULT MALES

Avg. Ambient Air Lead, μg/m^3	Total Absorbed Daily Dose[b]	
	Nonsmoker	Smoker
0.01	22.0	28.4
0.1	22.8	29.2
0.5	26.0	32.4
1.0	30.0	36.4
1.5	34.0	40.4
2.0	38.0	44.4
2.5	42.0	48.4
3.0	46.0	52.4
3.5	50.0	56.4
4.0	54.0	60.4
4.5	58.0	64.4
5.0	60.0	68.4

[a] Source: Battelle-Columbus Estimate

[b] Assumptions:

(1) Dietary intake of 254 μg Pb/day (suggested as intake level of young adult male in FDA Heavy Metals in Foods Survey (U. S. Department of Health, Education, and Welfare, 1977).

(2) Consumption of 1.5 liters of water/day at average lead content of 20 μg/l (= 30 μg/day).

(3) Consumption of 1 pack of 20 cigarettes/day or 6.4 μg Pb/day for smokers.

result in the ingestion of soil and/or dust. Within a contaminated environment, hand-to-mouth activity coupled with immature dieting habits (i.e., retrieval of food from dusty surfaces or soil, and subsequent consumption), provide a major source of ingested lead for young children. Day, et al., (1975) estimated that under average urban conditions, after 30 minutes of normal playground activity, 5 to 50 mg. of dirt from a child's hands can be transferred to a typical "sticky sweet". A small child playing in dirt easily ingests 10 mg. of soil/dust with each episode of hand-to-mouth activity. A conservative estimate of 10 hand-to-mouth activities a day would result in the ingestion of 100 mg. of soil/dust per day (Lepow, et al., 1975). One hundred mg. of ingested soil/dust per day would seem to be an approximate, conservative assumption for the average three year old on the empirical observations of Lepow, et al.

Pica, especially for paint, can also be a very important source, however, this habit is not characteristic of the majority of young children and, therefore, will be considered as a special case. Ingestion of non-food items is so variable that averages are not estimable for children with pica. Estimating average lead intakes from unintentional ingestion of non-food items (i.e., dust and dirt) among non-pica children is even difficult due to the apparent high variability in lead content of dust and dirt on environmental surfaces.

These cautions notwithstanding, estimates of the relative contribution of various media to lead absorption (dust/dirt, food, water and air) can be made for non-pica children given certain reasonable assumptions. Estimates based on three types of ambient air conditions and four different concentrations of lead in dust and dirt for a 3 year old child are given in Table 8.30. Daily intakes of food (1340 g/d) and daily dietary lead intake are based on figures from FDA's Total Diet Studies described in detail in Section 8.6.7. The gastrointestinal absorption rates of 41.5 used for food is suggested by Ziegler, et al., (1978). In the absence of estimated absorption rates for lead in water, the same percentage as for food is assumed to apply. Estimates of daily intake of water (1.4 ℓ) is based on reference man studies (ICRP, 1959) and is probably a liberal estimate since fruit juices and beverages made with water are included in FDA's analyses as foods. The level of 20 μg Pb/ℓ of water was selected as a value which would not be exceeded by more than a small percentage of households (see Section 8.3.2). Three different ambient air conditions are examined, since the relative contribution of other media varies as a function of air lead exposure. The levels selected to represent rural conditions (0.11 μg/m^3) and urban conditions (0.89 μg/m^3) appear realistic in view of the levels presented in Section 8.2.1. The levels selected for lead concentrations in dust and dirt (800, 1600, 2400, and 3000 ppm) were selected in order to include the range of mean values reported from samples taken from varying indoor and outdoor environments. The specific studies are detailed in Section 8.4.3 and indicated in footnote "k" of Table 8.30. It is believed that these concentrations are more realistic than use of the 11,000 ppm dust lead concentration reported by Lepow, et al., (1975), which has been used in some estimations. In support of this, Lepow, et al., actually found 2,400 ppm lead in the dust and dirt sampled directly from the children's hands. As noted earlier, it was assumed that absorption from dust and dirt is only three-quarters of that of the lead contained in food, or about 30-31 percent.

As shown in Table 8.30, food contributes a significant quantity of daily absorbed lead. Depending on the concentration of lead present in dust and dirt, food ranks either first or second (behind dust/dirt) as a source of lead body burden. The absolute contribution of water and air is fairly constant and low over the three environmental conditions depicted. Water contributes approximately 11.6 μg Pb/day and air, from 0.21 to 2.8 μg Pb/day at air lead levels up to the ambient air quality standard. The contribution from dirt and dust varies by a factor of four or more, (in the example from 24.9 to 99.5 μg Pb/day) depending on the assumptions made about the lead content of ingested dust and dirt. Consequently, it appears that in more heavily contaminated environments where lead in housedust and outdoor dirt/dust averages over approximately 1535 ppm, this source will provide equal or greater input than food to the "lead body burden of non-pica young children." This statement should, however,

TABLE 8.30 THE RELATIVE CONTRIBUTION OF VARIOUS MEDIA TO LEAD INTAKE AND ABSORPTION IN 3 YEAR-OLD CHILDREN

	Media	Daily Intake	Lead Concentration	Daily Lead Intake, μg/day	Percent absorbed	Daily Lead Absorption, μg/day
Rural 0.11μg/m^3	Food	1340 g[a]	0.1-0.2 ppm (variable)	115[b]	41.5[c]	47.7
	Water	1.4ℓ[d]	20μg/ℓ[e]	28	41.5[f]	11.6
	Air	4.7 m^3[g]	0.11μg/m^3	0.52	40[h]	0.21
	Dust/dirt	100 mg[i]	800 ppm[k]	80	31.1[j]	24.9
			1600 ppm	160	"	49.8
			2400 ppm	240	"	74.6
			3200 ppm	320	"	99.5
Urban 0.89μg/m^3	Food	1340g	0.1-0.2 ppm (Variable)	115	41.5	47.7
	Water	1.4ℓ	20μg/ℓ	28	41.5	11.6
	Air	4.7m^3	.89μg/m^3	4.2	40	1.7
	Dust/dirt	100 mg	800 ppm	80	31.1	24.9
			1600 ppm	160	"	49.8
			2400 ppm	240	"	74.6
			3200 ppm	320	"	99.5
At Ambient Air Quality Standard	Food	1340 g	0.1 - 0.2 ppm (Variable)	115	41.5	47.7
	Water	1.4ℓ	20μg/ℓ	28	41.5	11.6
	Air	4.7m^3	1.5μg/m^3	7.05	40	2.8
	Dust/dirt	100 mg	800 ppm	80	31.1	24.9
			1600 ppm	160	"	49.8
			2400 ppm	240	"	74.6
			3200 ppm	320	"	99.5

TABLE 8.30. (Continued)

Notes:

[a] 1340g intake figure based on averaging FDA Total Diet Studies estimates for 1-2 year old children (1290g/day) and 3-5 year old children (1390g/day). (See Table 8.33).

[b] 115µg/day, Battelle estimate based on average of FDA Total Diet Studies for male and female children 1-2 years of age (107µg/day) and children 3-5 years of age (123µg/day)

[c] 41.5 percent absorption rate based on Ziegler, et al., (1978) estimate.

[d] 1.4 liter per day intake based on 3 year old child as described by Drill, et al., (1978).

[e] 20µg/ℓ selected because at least 80 percent of households surveyed had tapwater below this level (McCabe, et al, 1976; U.S. Environmental Protection Agency, 1976).

[f] 41.5 percent absorption rate observed for foods by Ziegler, et al, assumed to apply to liquids as well.

[g] 4.7m^3/day estimated respiratory volume based on ICRP (1959)

[h] 40 percent absorption rate based on estimate by National Academy of Sciences, (1972).

[i] 100 mg/day intake based on estimate by Lepow, et al, (1974).

[j] 31.1 percent based on assumption that gastrointestional absorption of lead from dust and dirt is 75 percent as efficient as that from food.

[k] Estimates of lead present in dirt and dust are representative of the range of means observed by the investigators below:

600 ppm	indoor house duse (clean area)	Solomon and Natusch, 1977
1200 ppm	indoor house dust (aver. Boston and Manchester)	Kreuger, 1972
1200 ppm	outdoor play area dirt	Lepow, et al, 1974
1386 ppm	outdoor street dust	Hunt, et al., 1971
1658 ppm	outdoor street dust	Hunt, et al, 1971
2000 ppm	indoor house dust (core city Boston)	Kreuger, 1972
2344 ppm	outdoor street dust	Hunt, et al, 1971

be qualified by suggesting that the above estimate was based on ingestion of 100 mg/day of dust and dirt and an absorption rate of 31.1 percent. Although based on empirical studies (Lepow, et al., 1975), it is unknown whether this seemingly high level of dust and dirt ingestion is representative of normal children. (Lepow's estimate was based on observation of relatively disadvantaged urban children, all having blood leads over 40 μg/dl).

Table 8.31 shows the total daily lead absorption under the same environmental conditions described for Table 8.30. Once again, it is seen that food contributes approximately 30-57 percent of total absorbed lead. Likewise, dust/dirt contributes approximately 29-63 percent of total absorbed lead. The relative contribution of food and water vary inversely with lead concentration in dust and dirt. The contribution of air appears rather low and uniform, however, it should be realized that lead in air (along with weathering of paint, etc.) contributes to dust/dirt lead. Therefore, the entries in the air columns probably underestimate the full effects of lead in ambient air.

8.6.6 Recommendations Concerning Safe Levels of Dietary Lead

No evidence exists at the present time of any effect of lead as a beneficial trace element in human nutrition. Various estimates have been reported in Section 8.6.4 of the total human exposure to lead from food sources.

Data from the Total Diet Studies and the Heavy Metals in Foods Survey provide an estimate of daily lead intake for an American adult between 90-254 μg/day (U. S. Department of Health, Education, and Welfare, 1977). Consensus from other types of studies places intake in the range of 100-400 μg lead/day (National Academy of Sciences, 1972). This intake is well below that which produces toxicity in adults. Three hundred micrograms of lead per day has been found to be a level of ingestion at which positive balance does not occur in adults (Kehoe, 1961; U. S. Department of Health, Education, and Welfare, 1975). The World Health Organization (WHO) has recommended 3.0 milligrams per week (429 μg/day) as an acceptable total level of intake for adults. An intake of two hundred fifty four μg/day reported by the FY 1974 Heavy Metals in Foods Survey is 59 percent below this recommendation. Sufficient variability in intake levels shown by balance studies and institutional studies is suggestive of the possibility that some segments of the population may be consuming lead at levels much nearer Maximum Permissible Daily Intake (MPDI) of 429 μg. No population-based studies are available to confirm or deny this speculation, however. It may be concluded that present levels of dietary intake among the general population do not exceed maximum permissible intake as established by WHO but the margin of safety between present average levels of intake and the MPDI is not large. Certain segments of the population, particularly those exposed to substantial quantities of airborne lead (especially from occupation), receive additional ingested lead from air, and are possibly consuming lead at or above the MPDI of 429 μ/day.

Studies aimed at determining daily dietary lead intake of normal, non-pica children include studies of replicate diets, Total Diet Studies (FDA's Total Diet Studies and Heavy Metals in Food surveys), institutional diet studies and estimates based on fecal lead excretion.

TABLE 8.31. TOTAL DAILY LEAD ABSORBED BY 3 YR-OLD NON-PICA CHILD AND PERCENTAGE ATTRIBUTABLE TO VARIOUS MEDIA UNDER VARYING ENVIRONMENTAL CONDITIONS[a]

	Rural Conditions Air Lead = 0.11 $\mu g/m^3$					Urban Conditions Air Lead = 0.89 $\mu g/m^3$					Ambient Air Quality Standard Air Lead = 1.5 $\mu g/m^3$				
	Total Daily Lead Absorption under Various Levels of Lead in Dust and Dirt, μg[b]	Percent Contribution of Various Media				Total Daily Lead Absorption under Various Levels of Lead in Dust and Dirt, μg[b]	Percent Contribution of Various Media				Total Daily Lead Absorption under Various Levels of Lead in Dust and Dirt, μg[b]	Percent Contribution of Various Media			
Lead Conc. in Dust/Dirt, ppm		Dust/ Dirt	Food	Water	Air		Dust/ Dirt	Food	Water	Air		Dust/ Dirt	Food	Water	Air
800	84.4	29	57	14	<1	85.9	29	55	14	2	87.0	29	55	13	3
1600	109.3	46	44	10	<1	110.8	45	43	10	2	111.9	45	43	10	2
2400	134.1	56	36	8	<1	135.6	55	35	9	1	136.7	55	35	8	2
3000	159.0	63	30	7	<1	160.6	62	30	7	1	161.6	62	30	7	1

Notes: [a] See Table 8.30 for assumptions underlying these calculations.

[b] Sum of Column 6 of Table 8.30 for each of 4 lead levels in dust and dirt (i.e., : 800, 1600, 2400 and 3,000 ppm)

Kolbye, et al.,(1974) cited a dietary survey of 333 infants in the United States, aged 1 to 12 months, in which the estimated dietary lead intake was 93 plus or minus 36 micrograms per day. Similarly, the U. S. Department of Health, Education and Welfare (1975) has provided the following estimates for infants and toddlers through 2 years of age:

Age	Estimated μg/day Dietary Lead Intake
2 months	79
6 months	63
1 year	100
2 years	115

These estimates given above are based on survey data as well as on other sources discussed in more detail in the FDA report (U. S. Department of Health, Education, and Welfare, 1975). Based on examination of various surveys and methods, the general consensus is that normal children aged 1-3 consume, approximately, 100-130 μg of lead per day (Chisholm and Harrison, 1956; Barltrop and Killala, 1967; Mitchell and Aldous, 1974; Mahaffey, et al., 1975; U. S. Department of Health, Education, and Welfare, 1977).

The most significant inference to be drawn from these figures is that children receive a much higher dietary dose of lead than adults on a per weight basis. One survey showed that a diet consisting mostly of infant foods supplied 120 μg lead per day for a 6 month old child. As the child gets older and the diet consists more and more of adult table foods, lead intake actually decreases slightly (97-100 μg/day) due to the lower average lead content of adult foods. (Mahaffey, et al., 1975). If 200 μg/day is accepted as the average adult intake, then children under 2 years old receive as much as 1/2 of the adult dose.

The greater lead intake of infants, compared with adults, on a unit-body-weight basis, probably is related to infant's higher caloric and water requirements. It is not known whether the apparent greater intake is accompanied by a correspondingly greater total output; the critical balance studies have not been conducted, as they have in adults (National Academy of Science, 1972). Several studies do provide evidence of a higher rate of absorption from the alimentary tract in children. According to King (1971) and Alexander, et al., (1973), absorption rates in infants and young children may be as high as 50 percent compared to 5-10 percent for adults. Ziegler's recent (1978) studies indicating an average of 41.5 percent absorption confirm this high absorption for young children. Thus, the higher dietary dosage may take on added significance when coupled with a higher absorption rate.

Few official recommendations exist for acceptable lead intake levels in children. King (1971) reported that an ad hoc committee of the Department of Health, Education, and Welfare has suggested 300 μg/day for all sources as a maximum permissible intake for children. However, this recommendation was made before it was established that children absorb greater amounts of lead from the diet than adults. Lin-Fu (1972) estimated that the quantity of lead producing toxicity in children to be about 1000 µg/day for children 4-6 months of age. Barltrop (1972) has recommended a maximum permissible daily intake (MDPI) of 93-180 µg/day.

The mean intake estimated by the FDA Heavy Metals in Foods survey is below the 300 µg/day maximum permissible total intake proposed by King (1971). Data from the same survey (U. S. Department of Health, Education, and Welfare, 1975) have shown reason to doubt that lead in foods is normally distributed (i.e. there are substantially more high intakes than would be expected on the basis of a normal distribution). Occasional intakes of very high levels of lead in foods can and do occur. These occasional high findings greatly affect the estimated mean intake and if they can be reduced, the average exposure will substantially decrease. Also, it should be re-emphasized that the estimated dietary dosages do not apply to children with pica, who in eating a single chip of leaded paint, ingest many times the recommended maximum daily dose.

8.6.7 Effects of Reducing Adventitious Lead in Canned Foods on Dietary Lead Intake

This section describes the methods developed to estimate the effects on lead intake of reducing adventitious lead in canned foods. Estimates of current daily dietary lead intake are developed for various age-sex groups. These estimates are based on inventorying consumption of specific food commodities, determining the average amount of lead present in each commodity, and calculating daily lead intake by constructing a typical daily diet for various demographic groups. The amount of lead intake attributable to specific commodities/groups is tabulated in order to pinpoint those foods which contribute most significantly to total daily intake. (These tabulations do indeed verify that canned foods supply inordinate amounts of lead per unit.) The reduction in dosage, net and percentage, which would occur in the event of reducing lead in canned foods by one-half or two-thirds, is calculated for the various age-sex groups for which dietary data are available. Selection of these two reduction levels was based on two assumed possibilities. The maximum reduction of two-thirds is, essentially, equivalent to the total elimination of adventitious lead in canned food (since two-thirds of the total lead content is estimated to result from lead pickup). A less rigorous reduction assumes that total elimination of adventitious lead from canned food may not be practical, but that a 75 percent reduction in lead pickup can be achieved, equivalent to a 50 percent (0.75 x 0.667) overall reduction. For technical and economic reasons developed in Chapter 9, the 50 percent overall reduction is probably the more realistic case.

8.6.7.1 Methods for Deriving Food Consumption Levels--

Data from the Household Food Consumption Survey (from the U. S. Department of Agriculture, Agriculture Research Service, 1966) were modified to produce the data in Tables 8.32 to 8.36. The manipulations and assumptions made, deriving the information presented here from the published reports, are described below.

TABLE 8.32 ESTIMATED LEAD CONTENT OF VARIOUS FOODS

Food Class	Specific Food Commodity	Lead Content ppm	Basis for Estimate
Milk	Milk, fresh	0.021	FDA (1975b)
	Milk, processed, canned	0.12	FDA (1975a)
	Milk, processed, other	0.07	Reith,et al., (1974)
	Cream	0.068	FDA (1975b)
	Cheese and mixtures	0.12	MAFF (1975)
	Eggs	0.174	FDA (1975b)
Meat	Beef, canned	0.75	MAFF (1975)
	Beef, other	0.2	MAFF (1975)
	Pork, canned	0.13	Reith,et al., (1974)
	Pork, other	0.15	FDA (1975b)
	Poultry	0.127	FDA (1975b)
	Fish, canned	0.500	FDA (1975a)
	Fish, other	0.500	FDA (1975a)
	Other and mixtures, canned	0.28	CMI-NCA (1977b)
	Other and mixtures, other	0.198	FDA (1975b)
Nuts and Oils	Legumes and nuts	0.26	FDA (1975a)
	Fats and oils	0.013	FDA (1975b)
Breads	Bread	0.084	FDA (1975b)
	Other baked goods	0.10	Bogen (1972)
	Cereals, pastes	0.107	FDA (1975b)
Sugar Products	Sugar	0.031	FDA (1975b)
	Candy, jam, etc.	0.07	Reith,et al., (1974)
Beverages	Tea	0.20	Reith,et al., (1974)
	Coffee	0.07	Reith,et al., (1974)

TABLE 8.32 (Continued)

Food Class	Specific Food Commodity	Lead Content ppm	Basis for Estimate
Beverages (cont'd)	Cola, canned[a]	0.15	Ewing (1978)
	Cola, other[b]	0.08	Ewing (1978)
	Fruit drinks, canned	0.251	FDA (1975b)
	Fruit drinks, other	0.03	CMI-NCA (1977b)
	Other soft drinks, canned	0.02	Reith,et al., (1974)
	Other soft drinks, other	0.02	Reith,et al., (1974)
	Beer, canned	0.17	Reith,et al., (1974)
	Beer, other	0.10	Schroeder (1974)
	Wine	0.35	Schroeder (1974)
	Other alcoholic drinks	0.05	Reith,et al., (1974)
Tomatoes and Vegetables	Tomatoes, canned	0.710	FDA (1975b)
	Tomatoes, other	0.08	CMI-NCA (1977a)
	Tomato juice, canned	0.338	FDA (1975b)
	Citrus, canned[c]	0.09	CMI-NCA (1977b)
	Citrus, other	0.04	FDA (1975a)
	Citrus juice, canned	0.135	FDA (1975b)
	Citrus juice, other[d]	0.02	CMI-NCA (1977a)
	Dark green vegetables, canned[e]	0.44	CMI-NCA (1977b)
	Dark green vegetables, other	0.05	FDA (1973)
	Deep yellow vegetables, canned[e]	0.44	CMI-NCA (1977b)
	Deep yellow vegetables, other[e]	0.09	CMI-NCA (1977a)
Potatoes and Fruits	Potatoes, canned[f]	0.10	FDA (1975b)
	Potatoes, other	0.05	FDA (1975b)
	Other vegetables, canned[g]	0.48	CMI-NCA (1977b)
	Other vegetables, other	0.11	FDA (1975a)

TABLE 8.32 (Continued)

Food Class	Specific Food Commodity	Lead Content ppm	Basis for Estimate
Potatoes and Fruits (cont'd)	Other vegetable juice, canned	0.215	FDA (1975b)
	Other vegetable juice, other	0.04	CMI-NCA (1977b)
	Other fruits, canned	0.346	FDA (1975b)
	Other fruits, other	0.04	FDA (1975a)
	Other fruit juice, canned[h]	0.09	CMI-NCA (1977b)
	Other fruit juice, other	0.04	CMI-NCA (1977a)

[a] For "canned" vs. "other" in beverages, only lead-soldered cans were considered as canned since this is the basis for differentiation. Cans which do not leach lead are treated essentially as bottled products.

[b] Assumed value of 1/2 of the canned cola lead content.

[c] Based on canned grapefruit juice.

[d] Based on fresh orange juice.

[e] Based on mean of vegetables in the category.

[f] Assumed value of twice that of fresh.

[g] Based on corn.

[h] Based on apple and grapefruit juices.

TABLE 8.33 FOOD CONSUMPTION IN GRAMS PER DAY FOR VARIOUS AGE-SEX GROUPS

Food Class & Substance	Male/Female, Under 1 Year	Male/Female, 1-2 Years	Male/Female, 3-5 Years	Male/Female, 6-8 Years	Male, 15-17 Years	Female, 15-17 Years	Male, 20-34 Years	Female, 20-34 Years	Male, 55-64 Years	Female, 55-64 Years
MILK										
Milk, fresh	515.04	438.08	376.66	398.12	444.74	283.42	235.32	150.96	150.22	111.74
Milk, processed, canned	47.41	40.33	34.67	36.65	40.94	26.09	21.67	13.90	13.83	10.29
Milk, processed, other	133.55	113.59	97.67	102.93	115.32	73.49	61.02	39.14	38.95	28.97
Cream	18.0	23.0	31.0	40.0	49.0	34.0	33.0	30.0	39.0	35.0
Cheese and mixtures	1.0	5.0	4.0	5.0	11.0	8.0	12.0	11.0	15.0	14.0
Eggs	17.0	28.0	23.0	23.0	42.0	25.0	55.0	27.0	51.0	33.0
MEATS										
Beef, canned	0.08	0.26	0.37	0.42	0.83	0.64	1.21	0.71	0.89	0.59
Beef, other	6.92	23.74	33.63	37.58	74.18	57.36	108.79	63.29	80.11	53.41
Pork, canned	0.17	1.18	1.68	1.85	3.44	2.35	4.12	2.25	3.82	2.06
Pork, other	3.83	26.82	38.32	42.15	78.56	53.65	93.88	51.75	87.18	46.94
Poultry	3.0	11.0	16.0	25.0	27.0	18.0	32.0	21.0	28.0	25.0
Fish, canned	0.0	0.69	1.16	1.62	2.31	2.08	3.23	2.08	4.16	2.08
Fish, other	0.0	2.31	3.85	5.38	7.69	6.92	10.77	6.92	13.84	6.92
Other and mixtures, canned	4.42	4.82	4.02	5.36	9.11	6.57	10.59	6.43	6.83	5.90
Other and mixtures, other	29.58	33.18	28.98	36.64	62.89	46.43	74.41	46.57	55.17	44.10
NUTS & OILS										
Legumes and nuts	13.0	16.0	28.0	36.0	47.0	29.0	13.0	24.0	25.0	17.0
Fats and oils	2.0	12.0	18.0	22.0	39.0	22.0	15.0	23.0	35.0	24.0
BREADS										
Bread	5.0	39.0	63.0	77.0	135.0	79.0	120.0	71.0	107.0	67.0
Other baked goods	4.0	29.0	43.0	54.0	85.0	59.0	77.0	52.0	75.0	51.0
Cereals and pastes	48.0	75.0	83.0	84.0	93.0	65.0	84.0	75.0	73.0	52.0

TABLE 8.33 (Continued)

Food Class & Substance	Male/Female, Under 1 Year	Male/Female, 1-2 Years	Male/Female, 3-5 Years	Male/Female, 6-8 Years	Male, 15-17 Years	Female, 15-17 Years	Male, 20-34 Years	Female, 20-34 Years	Male, 55-64 Years	Female, 55-64 Years
SUGAR PRODUCTS										
Sugar	1.0	8.0	12.0	14.0	13.0	10.0	17.0	13.0	17.0	9.0
Candy, jam, etc.	9.0	22.0	31.0	33.0	46.0	31.0	27.0	22.0	30.0	22.0
BEVERAGES										
Tea	2.0	18.0	33.0	36.0	91.0	76.0	148.0	119.0	100.0	109.0
Coffee	0.0	2.0	5.0	4.0	59.0	65.0	422.0	400.0	558.0	522.0
Cola, canned	0.12	2.13	3.47	4.50	8.91	7.5	7.16	5.97	2.34	2.03
Cola, other	2.28	38.74	63.24	82.04	162.38	136.74	130.47	108.82	42.74	37.04
Fruit drinks, canned	0.04	0.63	1.03	1.35	2.65	2.23	2.13	1.78	0.70	0.61
Fruit drinks, other	0.68	11.54	18.84	24.52	48.37	40.73	38.86	32.41	12.73	11.03
Other soft drinks, canned	0.05	0.78	1.27	1.65	3.26	2.75	2.62	2.19	0.86	0.74
Other soft drinks, other	0.83	14.18	23.15	30.03	59.44	50.05	47.76	39.87	15.64	13.56
Beer, canned	0.0	0.0	0.0	0.0	0.0	0.0	6.15	1.31	4.12	0.68
Beer, other	0.0	0.0	0.0	0.0	0.0	0.0	112.17	23.92	75.05	12.37
Wine	0.0	0.0	0.0	0.0	0.0	0.0	7.34	1.57	4.91	0.81
Other alcoholic drinks	0.0	0.0	0.0	0.0	0.0	0.0	10.34	2.20	6.92	1.14
TOMATOES & VEGETABLES										
Tomatoes, canned	0.23	2.71	2.94	4.52	6.56	5.65	8.59	7.22	6.78	7.01
Tomatoes, other	0.56	6.71	7.27	11.18	16.21	13.98	21.24	17.90	16.77	17.33
Tomato juice, canned	0.21	2.58	2.79	4.30	6.23	5.37	8.17	6.88	6.45	6.67
Citrus, canned	0.21	0.46	0.52	0.53	0.72	0.59	0.64	0.52	0.64	0.73
Citrus, other	13.06	28.48	32.04	32.64	44.51	36.19	39.16	35.65	39.16	45.10
Citrus juice, canned	4.36	9.53	10.72	10.92	14.89	12.11	13.10	10.92	13.10	15.09
Citrus juice, other	4.37	9.53	10.72	10.92	14.89	12.11	13.10	10.92	13.10	15.09
Dark green vegetables, canned	0.31	0.61	0.46	0.77	0.92	1.22	1.07	1.22	1.22	1.07
Dark green vegetables, other	1.69	3.39	2.54	4.24	5.08	6.78	5.93	6.78	6.78	5.93

TABLE 8.33 (Continued)

Food Class & Substance	Male/Female, Under 1 Year	Male/Female, 1-2 Years	Male/Female, 3-5 Years	Male/Female, 6-8 Years	Male, 15-17 Years	Female, 15-17 Years	Male, 20-34 Years	Female, 20-34 Years	Male, 55-64 Years	Female, 55-64 Years
TOMATOES & VEGETABLES (continued)										
Deep yellow vegetables, canned	1.30	0.65	0.65	0.76	0.97	0.65	1.08	0.74	1.40	1.08
Deep yellow vegetables, other	10.70	5.35	5.35	6.24	8.03	5.35	8.92	6.26	11.60	8.92
POTATOES & FRUITS										
Potaotes, canned	0.10	0.58	0.73	0.90	1.55	0.85	1.63	1.06	1.29	0.82
Potatoes, other	5.90	33.42	42.27	52.10	89.45	49.15	94.37	54.94	74.71	47.18
Other vegetables, canned	14.28	13.71	13.71	16.28	26.57	21.14	32.28	22.90	28.58	22.85
Other vegetables, other	34.97	33.57	33.57	39.87	65.04	56.75	79.03	55.98	69.98	55.95
Other vegetable juice, canned	0.75	0.72	0.72	0.85	1.39	1.11	1.69	1.12	1.50	1.20
Other fruits, canned	21.99	19.63	19.83	22.18	23.16	22.18	18.85	12.46	21.79	18.06
Other fruits, other	81.72	72.97	73.70	82.46	86.11	82.46	70.05	46.42	81.0	67.13
Other fruit juice, canned	7.56	6.75	6.81	7.62	7.96	7.62	6.48	4.71	7.49	6.21
Other fruit juice, other	0.73	0.65	0.66	0.74	0.77	0.74	0.62	0.41	0.72	0.60

Report No. 1, entitled "Food Consumption of Households in the United States," gives detailed dietary information at the household level. The information is described for several different regions of the United States, degrees of urbanization (urban, rural farm, and rural non-farm) and household incomes. Data which have been aggregated across some or all of these parameters are tabulated as well.

Report No. 6 gives specific household food consumption data (in grams per day) for each food, separated according to its form (e.g. potatoes--fresh, potatoes--canned, etc.) This information is sufficient to derive, for any desired demographic group, a distribution of specific food forms for any given food. For example, assume a certain (demographic) household eats potatoes as follows: 60 g/day fresh, 27 g/day frozen, and 13 g/day canned. This is equivalent to a distribution of 60 percent fresh, 27 percent frozen, and 13 percent canned. This information is essential in merging this household-level data with individual-level data.

Report No. 11 (Food and Nutrient Intake of Individuals in the United States) is concerned with individual's diets. Demographic subdivisions are again provided, though in less detail, with the additional feature of diet specification by sex and age group. This provides the average grams per day intake for specific age-sex groups, divided/aggregated according to a variety of demographic descriptions.

This information is not, however, supplied in the same detail as the household data in Report No. 1. First, there is less specificity of the food types. For example, the category "tomatoes": includes all forms of whole tomatoes, tomato sauce, and tomato juice. The second difficulty involves failure to specify the form (i.e., fresh, frozen, or canned) of the product.

The household data were merged with the individual data in the following manner (as illustrated for tomatoes): Report No. 1 noted a household consumption of 1.09 g/day fresh whole tomatoes, 0.44 g/day canned tomatoes and sauce, and 0.42 g/day canned juice for an average income-region-urbanization household. This indicates that 78.5 percent of the tomatoes eaten were whole/sauce and 21.5 percent were in the form of juice. Furthermore, 28.8 percent of the whole/sauce products were canned while 100 percent of the juice was canned. Note that these proportions are based on the entire household's consumption.

Consultation of Report No. 11 states that a 20-to-34-year-old male (averaged over all incomes, urbanizations, and regions) eats 38 g of tomato products daily. Assuming his tomato intake is in the same relative proportions as the entire household, the above percentages are utilized. Thus, 78.5 percent of 38 g (29.83 g) is eaten daily as whole tomatoes or sauce. Of these 29.83 g, 28.8 percent, or 8.59 g, are canned and 21.24 g are not canned. Finally, 21.5 percent of the 38 g eaten daily is juice, all of which is canned (8.17 g day). This exemplifies the method employed for all the components of the diet.

In most cases, the basic nature of the product did not have to be extrapolated, e.g., meat did not have to be divided into beef, pork, etc., based on household averages since Report No. 11 supplied the meat and source. In all cases, however, the dichotomy of "canned" versus "not canned" required extrapolation. The only source used for extrapolation purposes, aside from the Household Food Consumption Survey Report No. 1, was a National Soft Drink Association (1970) report on canned beverages. This supplied the proportion of soft drinks and beer in bottles and several types of cans.

The potential sources of inaccuracy in the above method should be noted explicity. The food consumption survey data are quite old (1965-1966) and are in the process of being updated. In certain instances, where there are known changes in consumption patterns (e.g., the recent dramatic decline in the use of evaporated milk among infants), the changes of the past decade could be substantial. Another concern is the accuracy of assuming that all members of a household eat food items in the same proportions. For most adults, this may be reasonable, but deviations would be expected among extremely young children and perhaps among the elderly. These potential inaccuracies cannot be quantified but should be considered when evaluating the data presented. Unfortunately, the updated data being compiled by FDA are still in process of evaluation and verification, and have not yet been released, and were not available to this study.

8.6.7.2 Daily Dietary Lead Intakes from Different Foods for Various Age-Sex Groups--

Daily lead concentration and total daily consumption of various foods for several age-sex groups are presented in Tables 8.32 and 8.33. Lead content was multiplied by daily consumption in grams for each specific type of food to obtain average daily lead intake. Details of these calculations for each age-sex group are contained in Appendix A (Tables A-1 through A-10). These values were also calculated for the same age-sex group among low income households (incomes less than $3,000 per year). These values, however, were not sufficiently different from those based on the aggregate of all incomes to warrant their separate consideration.

Tables A-1 through A-10 also show the effects of reducing the lead in canned foods by one-half or two-thirds. Data for the various age-sex groups are summarized in Table 8.34. Distribution by age of amount of food ingested daily is shown in Figure 8.16 for males and in 8.17 for females. The reductions in daily lead intakes resulting from these decreases are summarized, by food group, in Table 8.35 for children and in Table 8.36 for adults.

As can be seen upon inspection of Table 8.34 and Figures 8.16 and 8.17, males 20-34 years old have the largest daily lead intake. The distribution of lead intake by age is similar in shape for males and females. However, the values for adult females are lower than those of the corresponding adult male age group. Also, the difference between female age groups is less than it is between corresponding male groups. The largest absolute reduction in daily lead intake is also in the male 20-34-year-old group, which is to be expected, due to the fact that this group originally had the largest intake. The largest

TABLE 8.34 DAILY DIETARY INTAKE OF LEAD FOR VARIOUS AGE-SEX GROUPS AND THE EFFECT OF REDUCING LEAD IN CANNED FOODS BY ONE-HALF OR TWO-THIRDS[a]

	Normal Daily Intake, µg	With Lead in Canned Food Reduced by 1/2		With Lead in Canned Food Reduced by 2/3	
		Daily Intake, µg	Reduction, Percent	Daily Intake, µg	Reduction, Percent
Male/Female, Under 1 year	75.42	63.47	15.8	60.04	20.4
Male/Female, 1-2 years	107.48	94.09	12.5	89.56	16.7
Male/Female 3-5 years	123.02	109.79	10.8	105.35	14.4
Male/Female, 6-8 years	143.60	127.80	11.0	122.20	14.9
Male, 15-17 years	222.64	200.95	9.7	193.73	13.0
Female, 15-17 years	168.03	150.63	10.5	143.71	14.6
Male, 20-34 years	273.52	247.73	9.4	239.26	12.5
Female, 20-34 years	190.36	173.94	8.6	168.46	11.5
Male, 55-64 years	236.03	216.12	8.4	209.06	11.4
Female, 55-64 years	183.02	166.22	9.2	154.46	15.6

[a] Source: Battelle-Columbus estimates.

TABLE 8.35. REDUCTION IN DAILY LEAD INTAKE FOR CHILDREN FROM VARIOUS FOOD CLASSES AFTER ELIMINATION OF 1/2 OR 2/3 OF THE LEAD IN CANNED FOODS[a]

Food Group	Proportion of Lead Removed	Age-Sex Groups							
		Males/Females, Under 1 Year		Males/Females, 1-2 Years		Males/Females, 3-5 Years		Males/Females, 6-8 Years	
		μg Reduction	Percent Reduction	μg Reduction	Percent Reduction	μg Reduction	Percent Reduction	μg Reduction	Percent Reduction
Milk	1/2	2.85	(9.7)	2.42	(8.3)	2.08	(8.2)	2.20	(8.1)
	2/3	3.79	(13.0)	3.23	(11.1)	2.77	(10.9)	2.93	(10.8)
Meats	1/2	0.66	(6.9)	1.03	(5.1)	1.12	(4.6)	1.43	(4.8)
	2/3	0.87	(9.2)	1.43	(7.2)	1.48	(6.1)	1.91	(6.4)
Nuts, Oils, Breads, and Sugar Products	1/2	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)
	2/3	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)
Beverages	1/2	0.01	(2.4)	0.25	(3.1)	0.41	(2.9)	0.53	(3.2)
	2/3	0.02	(3.2)	0.33	(4.2)	0.55	(4.0)	0.70	(4.3)
Tomatoes and Vegetables	1/2	0.78	(23.9)	2.34	(32.5)	2.51	(32.6)	3.43	(34.1)
	2/3	1.13	(35.0)	3.12	(43.3)	3.35	(43.5)	4.90	(48.7)
Potatoes and Fruits	1/2	7.62	(33.6)	7.36	(32.0)	7.14	(31.0)	8.22	(30.7)
	2/3	13.10	(42.3)	9.81	(42.6)	9.52	(41.3)	10.96	(40.9)
Totals	1/2	11.95	(15.8)	13.39	(12.5)	13.24	(10.8)	15.81	(11.0)
	2/3	15.38	(20.4)	17.91	(16.7)	17.67	(14.4)	21.40	(14.9)

[a] Source: Battelle-Columbus estimate

TABLE 8.36. REDUCTION IN DAILY LEAD INTAKE FOR ADULTS FROM VARIOUS FOOD CLASSES AFTER ELIMINATION OF 1/2 or 2/3 OF THE LEAD IN CANNED FOODS[a]

		Age-Sex Groups											
		Males, 15-17 Years		Females, 15-17 Years		Males, 20-34 Years		Females, 20-34 Years		Males, 55-64 Years		Females, 55-64 Years	
Food Group	Proportion of Lead Removed	µg Reduction	Percent Reduction	µg Reduction	Percent Reduction	µg Reduction	Percent Reduction	µg Reduction	Percent Reduction	µg Reduction	Percent Reduction	µg Reduction	Percent Reduction
Milk	1/2	2.46	(7.2)	1.31	(6.0)	1.30	(5.1)	0.84	(5.3)	0.83	(4.0)	0.72	(4.6)
	2/3	3.27	(9.6)	2.09	(9.6)	1.73	(6.9)	1.11	(7.1)	1.13	(5.4)	0.92	(5.9)
Meats	1/2	2.39	(4.7)	1.83	(4.8)	1.40	(2.2)	1.83	(4.7)	2.58	(4.6)	1.70	(4.6)
	2/3	3.18	(6.2)	2.44	(6.4)	2.41	(3.7)	2.44	(6.2)	3.44	(6.2)	2.26	(6.2)
Nuts, Oils Breads, and Sugar Products	1/2	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)
	2/3	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)	0.0	(0.0)
Beverages	1/2	1.03	(2.6)	0.87	(2.5)	1.35	(1.5)	0.80	(1.2)	0.32	(0.4)	0.29	(0.5)
	2/3	1.38	(3.4)	1.16	(3.4)	1.80	(2.0)	1.06	(1.6)	0.83	(1.1)	0.39	(0.6)
Tomatoes and Vegetables	1/2	4.83	(34.5)	5.62	(41.9)	8.46	(39.3)	4.93	(35.6)	4.99	(34.3)	5.14	(34.5)
	2/3	6.45	(46.0)	7.01	(52.2)	11.28	(52.3)	6.57	(44.5)	6.65	(47.5)	6.86	(46.1)
Potatoes and Fruits	1/2	10.97	(29.6)	9.80	(31.0)	11.66	(29.6)	8.04	(29.9)	11.19	(30.2)	9.06	(30.9)
	2/3	14.62	(39.5)	13.06	(41.3)	15.41	(39.2)	10.71	(40.0)	14.92	(40.2)	18.23	(62.1)
Totals	1/2	21.69	(9.7)	17.68	(10.5)	25.79	(9.4)	16.43	(8.6)	19.91	(8.4)	16.81	(9.2)
	2/3	28.91	(13.0)	24.60	(14.6)	34.26	(12.5)	21.90	(11.5)	26.97	(11.4)	28.56	(15.6)

[a] Source: Battelle-Columbus estimate

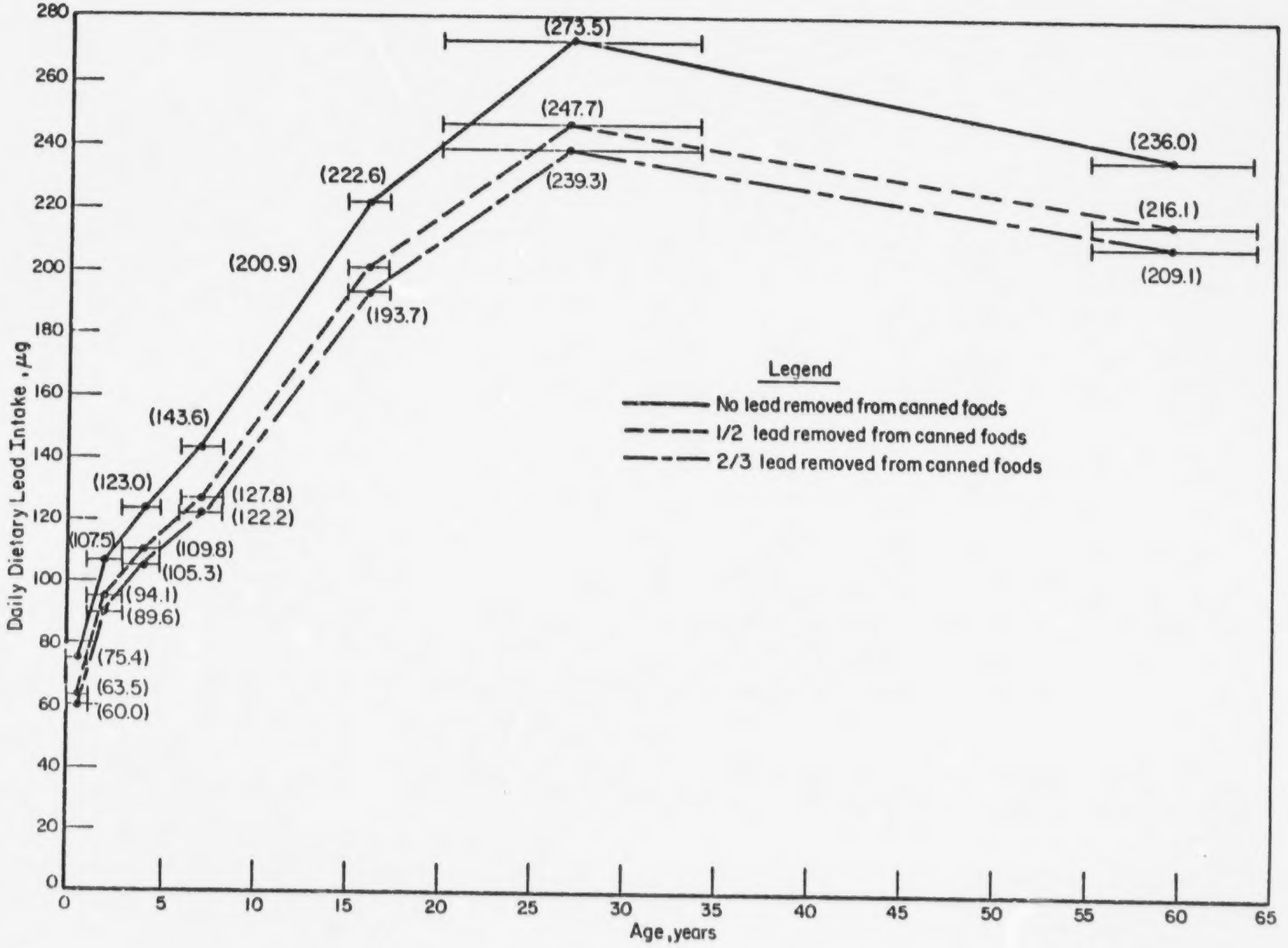


Figure 8.16 Daily dietary intake of males, by age

Source: Battelle-Columbus estimate

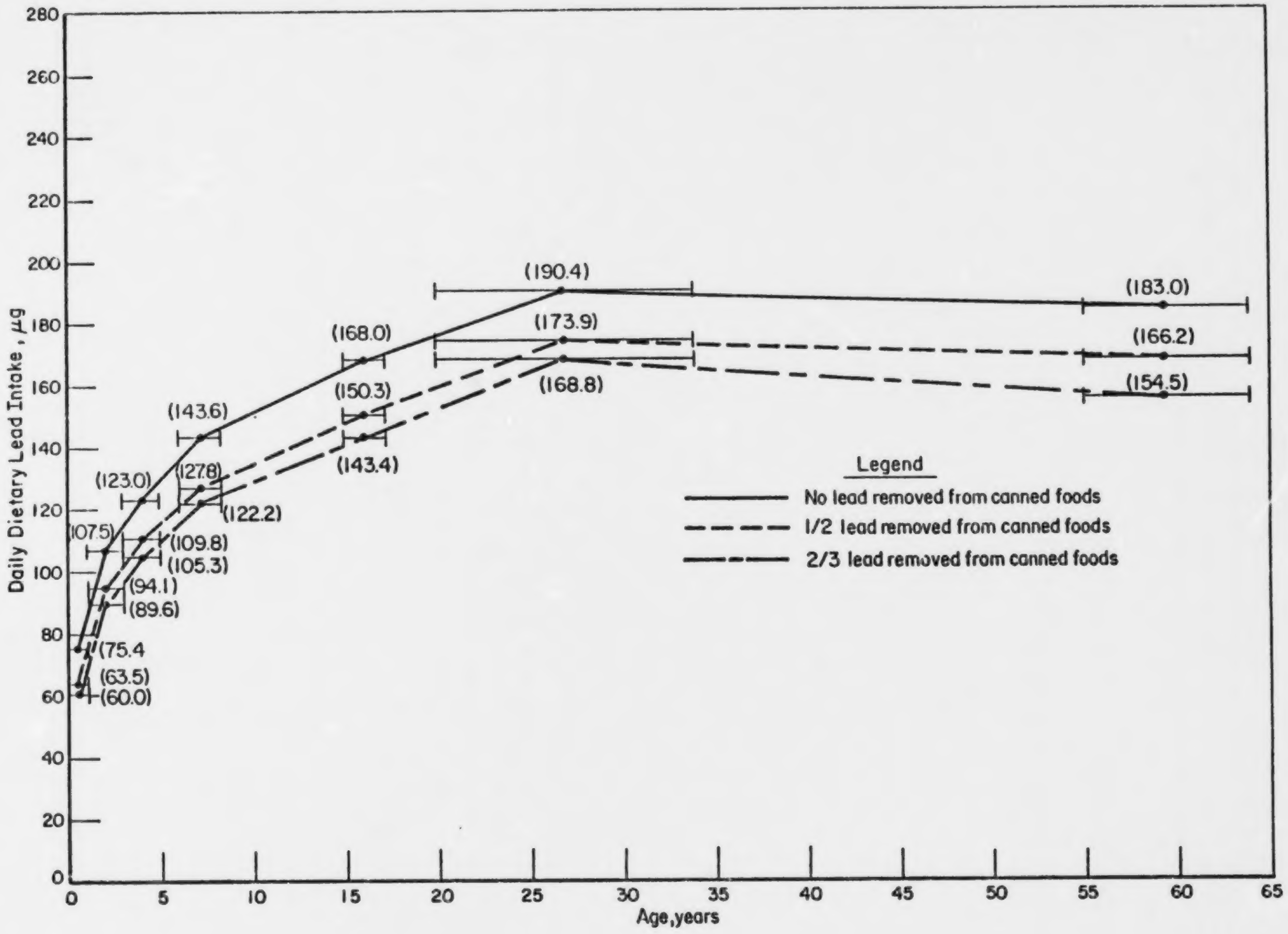


Figure 8.17. Daily dietary intake of females, by age

Source: Battelle-Columbus estimate

percentage reduction can be seen in children less than 1 year old.

Across all the age-sex groups, the food groups in which the largest reduction occurs are the "tomato and vegetable" and "potato and fruit" groups. However, the fact that these groups contribute only a small percentage of the total daily food intake must be considered. This somewhat diminishes the effect of the large percentage reduction within these groups.

8.7 LEAD UPTAKE/BLOOD-LEAD RELATIONSHIPS

8.7.1 Relationships of Blood-Lead Levels to Lead Exposure in Humans

Various studies indicate the body burden of lead accumulates until middle age and perhaps beyond (National Academy of Sciences, 1972); U. S. Environmental Protection Agency, 1977). Although the body burden is distributed throughout a number of organs and systems within the body, the largest portion is concentrated in the skeletal system. This is also where the accumulation occurs, producing a reservoir of lead in the body that is not readily exchangeable nor biologically available under normal conditions (National Academy of Sciences, 1972). Thus, the non-accumulating portion of the body burden of lead is more toxicologically significant than the lead reservoir in the skeletal system which gradually increases with age (U. S. Environmental Protection Agency, 1977). In contrast to the relatively nonexchangeable lead reserves in the skeleton, much of the exchangeable portion of the body burden is stored in the soft tissues such as the liver and kidney, which are perfused with blood. In consideration of this, the blood lead level is often used as an indicator of the concentration of lead in soft tissues. Blood lead is, therefore, considered to be a more useful parameter than total body burden, since it is thought to be representative of the exchangeable portion of the body burden. Moreover, blood is more easily accessible than tissue for biological monitoring of lead.

There have been numerous attempts to demonstrate a dose-response relationship between lead dosage received from various media and blood lead. Two main approaches have been used. The first assumes that the concentration of blood lead is proportional to the combined level of absorption via all the modes of exposure to lead (mainly air, food, and water). Next, the percentage of the total inhaled and ingested lead actually absorbed by the body is calculated under various assumptions regarding retention rates for various media. It follows that each source of exposure contributes to the concentration of blood lead in proportion to the amount absorbed from that source. (Bell, et al., 1978). In general, an adult ingests, approximately, 200-300 μg of lead per day from dietary sources. Assuming an intake of 250 μg of lead per day and 7.5 percent absorption of the dose, it follows that 18.75 μg/day would be absorbed from diet. From an urban atmosphere averaging 2.0 $\mu g/m^3$ of lead, an assumed respiratory intake of 20 m^3 per day and a retention rate of 37 percent, absorption of 14.8 μg of lead would occur. An additional 5-10 μg per day might be absorbed by those who smoke cigarettes. The contribution to absorbed dose from drinking

TABLE 8.37. GEOMETRIC MEAN AIR-AND BLOOD-LEAD VALUES FOR 11 STUDY POPULATIONS[a]

Community	Geom. mean air, lead, μg/m³	Blood lead, μg/dl		Sample Size
		Geom. mean	GSD	
Los Alamos, NM	0.17	14.9	1.28	185
Okeana, OH	0.32	15.6	1.39	156
Houston, TX	0.85	12.5	1.31	186
Port Washington, NY	1.13	15.4	1.28	196
Ardmore, PA	1.15	18.0	1.38	148
Lombard, IL	1.18	13.9	1.27	146
Washington, DC	1.19	19.2	1.26	219
Rittenhouse, PA	1.67	20.6	1.33	136
Bridgeport, IL	1.76	17.6	1.27	146
Greenwich Village, NY	2.08	16.6	1.28	139
Pasadena, CA	3.39	17.5	1.31	194

[a] Source: U.S. Environmental Protection Agency (1977); Calculated from data from Tepper and Levin (1975).

water amounts to, approximately, 3 μg/day, assuming intake of 1.5 liters of water at 20 μg/ℓ and 10 percent absorption. Thus, it appears that the relative contributions of dietary (including drinking water) and atmospheric sources to the blood lead level of a non-smoking urban adult are, approximately, 67 and 33 percent, respectively, (14.8 μg from air divided by 18.75 from diet and 3.0 μg/day from water) Ewing and Pearson, 1974; Environmental Health Resources Center, 1973; National Academy of Sciences, 1972. The proportion contributed by diet in children is probably closer to 75 percent (Environmental Protection Agency, 1977; Environmental Health Resources Center, 1973). Presumably, this is attributable to higher absorption rates of children. Ratios of ingested to absorbed lead may be as high as 0.5 (50 percent absorption) for infants as opposed to 0.05 to 0.10 observed among adults. Although empirical data are lacking, it is probable that the absorption ratio decreases gradually throughout childhood.

The second type of approach used to demonstrate this dose-response relationship is the comparison of lead exposure from diet and atmosphere with the blood lead level. Qualitatively, the concentration of blood lead increases as exposure from any or all sources increases. Studies have shown that this is not a constant ratio over a broad range of exposures, however. (U. S. Environmental Protection Agency, 1977; Bell, et al., 1978; World Health Organization, 1977). The relationship appears to be linear for low exposures and logarithmic for very high exposures (U. S. Environmental Protection Agency, 1977). In the lower ranges of atmospheric exposure, blood lead levels increase 0.6 to 2.0 μg/dl blood for every 1.0 μg per m^3 atmospheric lead (U. S. Environmental Protection Agency, 1977; Bell et al., 1978; World Health Organization, 1977). A similar quantitative relationship for dietary exposure is not well established. Various studies have shown increases in blood lead from 6 to 18 μg per dl blood for every 100 μg lead ingested daily; European data provide a more consistent picture of this relationship. U. S. Environmental Protection Agency (1977) indicates that each 100 μg Pb/day from diet contributes about 6 μg Pb/dl to blood lead. This relationship, in turn, implies that the mean blood lead level of American adults exposed to low levels of atmospheric lead (<1 μg/m^3 average annual mean) should be in the vicinity of 15 μg Pb/dl, assuming a total dietary intake of 250 μg/day:

$$(250/100)(6\ \mu g\ Pb/dl) = 15\ \mu g\ Pb/dl$$

Empirical observation confirms that this estimate closely approximates the mean blood lead values given for eleven study populations shown in Table 8.37.

8.7.2 Distributional Characteristics of Blood Lead Values in General Populations

Several studies have reported that evidence of adverse health effects (e. g., increased urinary ALA) can be detected at blood-lead levels above 40 μg/dl (Environmental Health Resource Center, 1973; U. S. Environmental Protection Agency, 1977; Tepper and Levin, 1975). The inhibition of aminolevulinate dehydratatase (ALA-D) can be detected at blood-lead levels as low as 10 μg/dl (U. S. Environmental Protection Agency, 1977). In evaluating the extent to which members of general populations exhibit blood-lead levels above these critical levels, it is essential to investigate the distribution of blood-lead levels in large samples of the general population unexposed to unusual sources of lead (i.e., normal populations).

It is reasonable to assume that even though average population blood-lead levels are well below the threshold level for adverse effects, a certain percentage of individuals may still be above this level depending upon the average and standard deviation of this distribution (Schubert, et al., 1967). Allowance for biological variability is a critical issue in: (1) setting standards for toxic substances based on avoidance of adverse health effects, (2) evaluating the effectiveness of such standards via biological monitoring, and (3) evaluating changes in the population parameters. Specifically, the critical question to be answered concerns the effect on high risk groups of, significantly, lowering the average population blood-lead level. Lowering the mean blood-lead level of the population should, theoretically, reduce the number of individuals above a specified critical level, provided that the standard deviation and the shape of the distribution remain unchanged.

In order to develop a method for predicting the response of blood-lead levels in a population to a specified reduction in lead intake, it is necessary to consider the distributional characteristics of blood lead values for various populations and to examine the statistical properties implied by the form of these distributions.

The distribution of blood-lead levels is most often described as being log-normal; that is, skewed to the right (U. S. Environmental Protection Agency, 1977; Tepper and Levin, 1972; Yankel, et al., 1977). The logarithm of a variable that is log-normally distributed will be normally distributed and will adhere to all the assumptions and rules of any other normally distributed variable (Schubert, et al., 1967; Aitchison and Brown, 1963). The median is a more useful measure of central tendency for a skewed distribution such as this than is the arithmetic mean. Since in the case of blood-lead distribution, the arithmetic mean will lie to the right of the median by an amount determined by the standard deviation, the geometric mean is a better estimate of the central tendency. Geometric Mean (GM) is defined as follows:

$$(GM) = \text{Exp} \sum_{i=1}^{n} (\ln x_i)/n]$$

The standard deviation of this statistic, the Geometric Standard Deviation, (GSD), is defined as follows:

$$(GSD) = \text{Exp} \{\sum_{i=1}^{n} [\ln(x_i) - \ln(GM)]^2/n-1\}^{1/2}$$

(Aitchison and Brown, 1963; Schubert, et al., 1967). In the following discussion and caluculations, the geometric statistics will be used rather than arithmetic ones.

8.7.3 Effects of Reducing Dietary Intake on Mean Blood Lead Levels

Control strategies, directed toward the elimination of lead added in canning, benefit, practically, the entire population, because consumption of canned products is so universal. Consequently, it follows that substantial reductions in dietary lead would produce reductions in blood lead proportional to current intake of canned products. Groups of consumers with higher than average consumption of canned products, e.g., infants, the poor, the institutionalized, the elderly and those living in remote areas might expect the greatest beneficial effect.

Changes, such as those projected, can also be thought of in terms of a shift to the left (downward) of the frequency distribution of blood-lead levels of a population. Providing: (1) baseline data are available for the population group of interest (i.e., geometric mean, GM, and geometric standard deviation, GSD) and, (2) the shape of the distribution can be assumed to remain constant, then estimates of the percentage of the population at or above any selected "biological threshold level" can be ascertained. Again, assuming that in the event of a reduction in dietary lead intake, the distribution curve shifts to the left with no change in shape, estimates of the percentage reduction in individuals at or above any desired level can be deduced. Moreover, if estimates of the size of the specific population group of interest are available from census or other sources, the percentage reduction can be expressed in terms of an absolute number of individuals. This provides an excellent criterion for judging the potential effectiveness of any anticipated program/limitation strategy, if the necessary data for such an analysis are available. Unfortunately, this is the exception rather than the rule, and such calculations are not possible for other limitation candidates. Even for dietary intake of lead, sufficient data are available only for the demographic group consisting of 20-34-year-old females. This group is used as a test case to demonstrate the approach (Section 8.7.3.2).

8.7.3.1 Approach to Estimation--

As suggested above, it appears that on the average for adults, diet contributes, approximately, 6 μg of lead/dl for each 100 μg of lead ingested in the diet. Assuming that this average might be applied to an hypothetical individual with a blood-lead level of 15 μg Pb/dl, it follows that if his diet contains 200 μg of lead per day, 12 μg/dl or 80 percent of his total blood-lead is accounted for by dietary sources, while the remaining 3 μg/dl or 20 percent comes from other sources, primarily, atmospheric lead (and, possibly, cigarettes).

In order to calculate the amount of reduction in the blood-lead level which would result from lowering dietary lead by a specific amount, it is necessary to know the actual amount of lead ingested daily and the amount of reduction achieved/desired. For example, if the same hypothetical individual with a blood-lead level of 15 μg/dl ingests 200 μg of lead daily and, subsequently, reduces his intake to 150 μg/day, the reduction in blood-lead resulting from the lowering of dietary intake can be predicted, using the 6 μg/dl per 100 μg intake relationship.

The reduced dietary intake, 150 μg/day would contribute 9.0 μg/dl to the blood-lead level to which the 3 μg/day contributed by air would be added (since it remains unchanged). Thus, a blood-lead level of 12.0 μg/dl might be expected on the reduced dietary lead regimen as opposed to the original value of 15 μg/dl. (To achieve the same 3 μg/dl reduction in blood-lead by reducing air lead rather than dietary lead, air lead would have to be reduced, approximately, 2.0 μg/m^3 below present levels (assuming each 1 μg/m^3 of lead in air contributes about 1-2 μg/dl to blood-lead). Since there are almost no NASN stations exceeding this value, it is apparent that, for practical purposes, reducing dietary intake is a more promising avenue to pursue.

Although the present example utilized a hypothetical individual case, similar calculations can be made for specific population groups (i.e., children under 5, women in childbearing years, etc.) using the actual (current) amounts of lead ingested and estimates of the reductions which can be effected for each group, using information regarding dietary lead intakes of various age-sex groups as well as net and percentage reductions under various technological options such as that presented in Table 8.34.

Another application of analysis of the frequency distribution of blood-lead levels in populations is in the determination of the proportion of the population whose blood lead values exceed a selected critical, or "threshold" level. For example, the percentage of a population with blood-lead above 40 μg/dl, regarded by many as a critical level, can be estimated using the logarithms of geometric means and standard deviations derived from empirical data (Aitchison and Brown, 1963; Schubert, et al., 1967; U. S. Environmental Protection Agency, 1977). Such a procedure allows prediction of the effects of any contemplated change in exposure level, provided that the effect can be expressed in terms of a change in the population mean. Starting from the baseline or current levels, the percentage of the population exceeding a critical level is determined. If the mean of that population changes by a given amount due to some intervention, the projected change in percent of the population above the selected critical level can be projected (assuming that the standard deviation of the distribution remains the same, a reasonable assumption).

8.7.3.2 Demonstration of the Technique in a Test Case--

Although the change in percentage above a critical level may be small, the absolute quantity of individuals shifting from above to below a critical threshold level can be quite large. It is expected that many individuals at or above critical levels will be shifted to safe "subcritical" levels as a result of lowering the mean blood-lead level for the population, as shown in the following example.

Several researchers have found that hematologic changes can be clearly seen at blood-lead levels around 25 μg/dl, especially in women and children, (Committee on Toxicology, 1976; U. S. Environmental Protection Agency, 1977; Environmental Health Resources Center, 1973). The effect of a reduction in lead intake on the percentage of the population group of women 20-34 years old with blood-lead values above 25 μg/dl will be calculated as a specific example. The geometric mean blood-lead level of this group has been estimated to be 16.6 μg/dl (Tepper and Levin, 1972; U. S. Environmental Protection Agency, 1977). Theoretically, the geometric standard deviation is constant at 1.31 μg/dl; this has been empirically verified in several cases (Yankel, et al., 1977; Environmental Protection Agency, 1977).

Using the 6 μg/dl per 100 μg dietary intake of lead, and the 190.4 μg/day intake for this population subgroup (Table 8.34), it can be calculated that 11.4 μg/dl of blood lead is derived from food, with the 5.2 μg/dl difference being attributable to air and other causes. This intake would be reduced to 168.5 μg/day if two-thirds of the lead in the canned food portion of the diet were eliminated (Table 8.34). The corresponding blood lead contribution from diet would be 10.1 μg/dl which, plus the 5.2 μg/dl contribution from non-dietary sources, would give a new mean blood-lead level of 15.3 μg/dl.

The percentage of the population with blood-lead values above 25 μg/dl can now be calculated for the two different means. In order to use the assumptions of a normal distribution, Z values must be calculated using logarithmic transformations of the geometric variables described above. The general formula for Z is as follows: $Z = (X - \mu)/\partial$, where μ is the mean, and ∂ is the standard deviation.

Percentages of population corresponding to any desired blood levels are easily calculated by computer. Data for the two populations of females 20-34 years of age, having blood-lead means of 16.6 μg/dl and 15.3 μg/dl, are used in the following example:

For GM = 16.6 μg/dl:

$$Z = [\ln(25\mu g/dl) - \ln(16.6\mu g/dl]/\ln(1.31\mu g/dl) = 1.52$$

For GM = 15.3:

$$Z = [\ln(25\mu g/dl) - \ln(15.3\mu g/dl/\ln(1.31\mu g/dl = 1.81$$

Figure 8.18 shows two curves describing distributions of blood lead levels in two hypothetical populations of women 20-34 years of age. The percentage plotted versus any given level represents the proportion of the population expected to have blood lead levels greater than or equal to that given value. Blood-lead levels in both populations were assumed to have lognormal distributions. For the first and second curves, the geometric means (GM) were taken to be 16.6 and 15.3, respectively; for both curves, the geometric standard deviation (GSD) was taken to be 1.31.

The proportions corresponding to the percentages plotted in the graph were computed as follows. Since p is the proportion of people having blood lead levels less than or equal to a given level x, 1 - p is the proportion having levels greater than x. Also, $p = \Pr(Z < z)$, where z is set equal to $(\ln(x) - \ln(GM)/\ln(GSD)$. Since the blood lead levels are lognormally distributed, this last expression has a standard normal probability distribution. Therefore, "p" values can be directly obtained from a table of cumulative normal distribution function values (Z-scores). The percentage values of interest were then computed as $(1 - p) \times 100$.

The curves in Figure 8.18 were plotted after using this method to compute percentages corresponding to blood lead levels (x) ranging from 15 to 45 for the two hypothetical human populations. Table 8.38 shows the actual percentages at

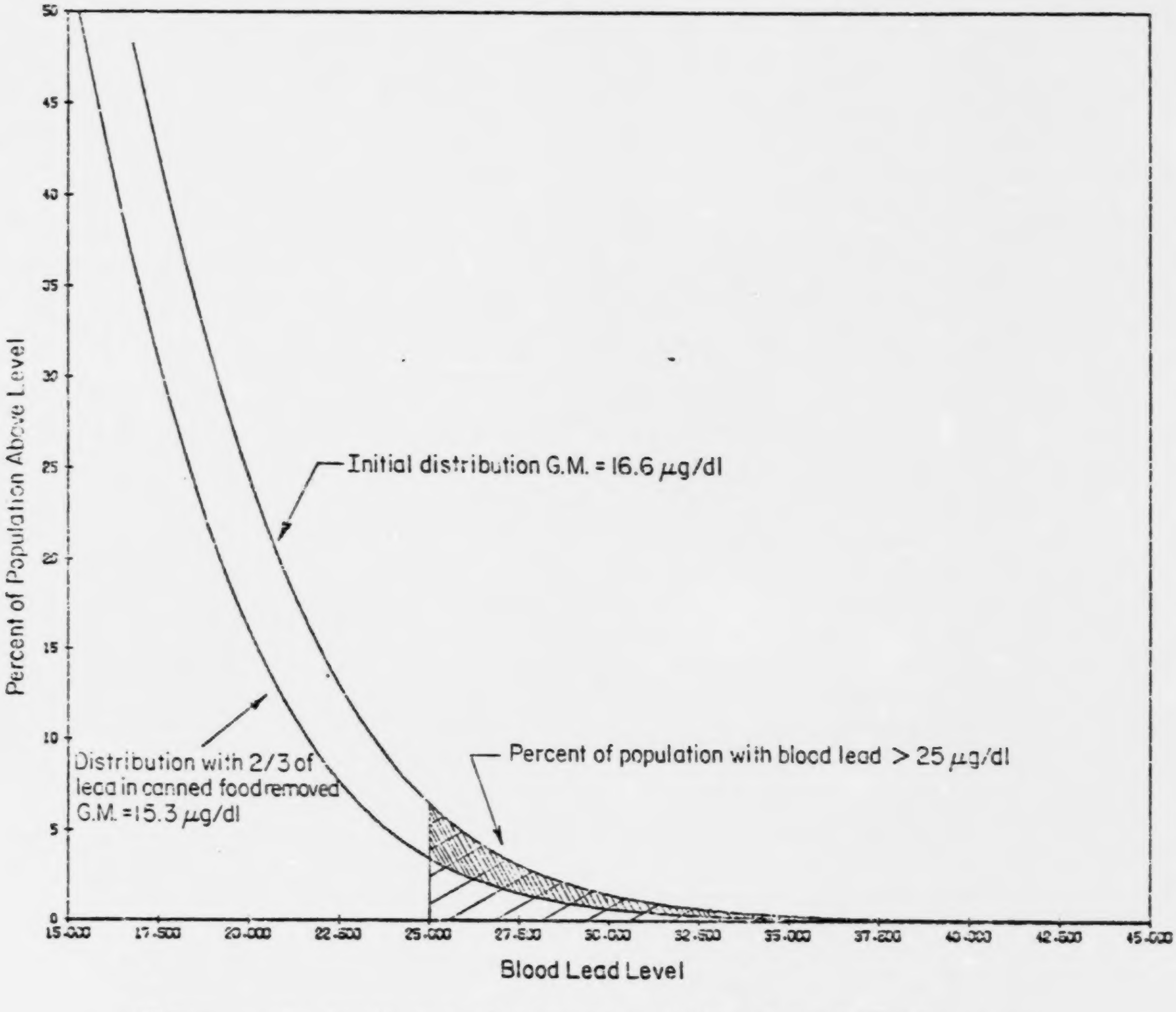


Figure 8.18. Predicted distribution of blood lead levels at two different mean blood levels for adult women, 20-34 years old.

Source: Battelle-Columbus estimate

TABLE 8.38. FREQUENCY DISTRIBUTION OF BLOOD LEAD VALUES IN TWO HYPOTHETICAL POPULATIONS OF ADULT FEMALES[a]

Blood Lead Level µg/dl	Percentage of Population at or above the Blood Lead Level Indicated in Column 1	
	Population 1[b]	Population 2[c]
15.0000	64.6297	52.9231
15.3000	61.8676	50.0000
15.6000	59.0991	47.1336
15.9000	56.3383	44.3361
16.2000	53.5988	41.6180
16.5000	50.8926	38.9881
16.8000	48.2312	36.4537
17.1000	45.6244	34.0204
17.4000	43.0812	31.6926
17.7000	40.6091	29.4730
18.0000	38.2144	27.3633
18.3000	35.9024	25.3641
18.6000	33.6773	23.4750
18.9000	31.5421	21.6946
19.2000	29.4991	20.0211
19.5000	27.5494	18.4516
19.8000	25.6936	16.9833
20.1000	23.9314	15.6124
20.4000	22.2620	14.3352
20.7000	20.6838	13.1475
21.0000	19.1952	12.0452
21.3000	17.7937	11.0238
21.6000	16.4767	10.0791
21.9000	15.2414	9.2066
22.2000	14.0847	8.4020
22.5000	13.0034	7.6612
22.8000	11.9941	6.9799
23.1000	11.0534	6.3543
23.4000	10.1780	5.7803
23.7000	9.3642	5.2545
24.0000	8.6089	4.7733
24.3000	7.9087	4.3333
24.6000	7.2602	3.9315
24.9000	6.6604	3.5649
25.2000	6.1062	3.2306
25.5000	5.5946	2.9262
25.8000	5.1228	2.6491
26.1000	4.6881	2.3971
26.4000	4.2880	2.1681
26.7000	3.9200	1.9601
27.0000	3.5818	1.7714
27.3000	3.2712	1.6002
27.6000	2.9862	1.4451
27.9000	2.7249	1.3045
28.2000	2.4854	1.1772
28.5000	2.2661	1.0621
28.8000	2.0653	.9579
29.1000	1.8817	.8637
29.4000	1.7139	.7786
29.7000	1.5605	.7017
30.0000	1.4204	.6322
30.3000	1.2925	.5696
30.6000	1.1758	.5130
30.9000	1.0694	.4619
31.2000	.9723	.4159
31.5000	.8839	.3744

TABLE 8.38 (Continued)

Blood Lead Level µg/dl	Percentage of Population at or above the Blood Lead Level Indicated in Column 1 Population 1[b]	Population 2[c]
31.8000	.8033	.3370
32.1000	.7300	.3033
32.4000	.6632	.2729
32.7000	.6024	.2456
33.0000	.5471	.2210
33.3000	.4967	.1988
33.6000	.4510	.1788
33.9000	.4094	.1608
34.2000	.3716	.1447
34.5000	.3372	.1301
34.8000	.3060	.1170
35.1000	.2777	.1052
35.4000	.2519	.0946
35.7000	.2285	.0851
36.0000	.2073	.0765
36.3000	.1881	.0688
36.6000	.1706	.0619
36.9000	.1547	.0557
37.2000	.1403	.0501
37.5000	.1272	.0450
37.8000	.1154	.0405
38.1000	.1046	.0364
38.4000	.0949	.0327
38.7000	.0860	.0295
39.0000	.0780	.0265
39.3000	.0707	.0238
39.6000	.0642	.0214
39.9000	.0582	.0193
40.2000	.0528	.0174
40.5000	.0478	.0156
40.8000	.0434	.0140
41.1000	.0393	.0126
41.4000	.0357	.0114
41.7000	.0324	.0102
42.0000	.0293	.0092
42.3000	.0266	.0083
42.6000	.0241	.0075
42.9000	.0219	.0067
43.2000	.0199	.0061
43.5000	.0180	.0055
43.8000	.0163	.0049
44.1000	.0148	.0044
44.4000	.0135	.0040
44.7000	.0122	.0036
45.0000	.0111	.0032

---

[a] See text for full description of how these data were generated.

[b] Geometric mean blood lead 16.6 µg/dl

[c] Geometric mean blood lead 15.3 µg/dl

or above a given blood level for each of the hypothetical populations.

In a population with geometric mean at 16.6 μg/dl, 6.55 percent of individuals would be expected to have blood lead levels at or above 25 μg/dl. On the other hand, in a population with geometric mean at 15.3, only 3.22 percent would be expected to have blood lead levels at or above 25 μg/dl. Had another critical blood lead value been selected, say 40 μg/dl, Table 8.38 shows that for the population with geometric mean of 16.6, over 5 percent of the population would be at or above 40 μg/dl while in the second population (geometric blood lead mean at 15.3) less than 2 percent would reach levels as high as 40 μg/dl.

Extending these results to real world conditions illustrates the dramatic effect of a small reduction (1.3 μg/dl) in the geometric mean blood level of a population. According to the 1970 census, there were 21,143,824 women between the ages of 20 and 34 in the United States (U. S. Bureau of Census, 1975); 1,384,920 of these would be expected to have blood-lead levels above 25 μg/dl. However, as a result of lowering the mean blood-lead level in this group as described above, the number with levels above 25 μg/dl can be reduced to 680,831. Obviously, the reduction in mean blood-lead level described above can have profound effects; it would lower by 1/2 the number of people in the range where evidence of hematologic changes begin to appear. Furthermore, since the highest fertility occurs among women of the 20-34 age group, additional benefits, in terms of protecting unborn children from undue lead exposure, would also be expected.

8.8 REFERENCES

Aitchison, J. and J. A. C. Brown. 1963. The Lognormal Distribution. Cambridge University Press.

Akland, G. G. 1976. Air Quality Data for Metals, 1970 through 1974, from The National Air Surveillance Networks, EPA-600/4-76-041, U.S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory, Reasearch Triangle Park, North Carolina. August.

Alexander, F. W., H. T. Delves, and B. E. Clayton. 1973. The Uptake and Excretion by Children of Lead and Other Contaminants. In: Proceedings, International Symposium, Environmental Health Aspects of Lead, Amsterdam, Oct. 2-6, 1972, D. Barth, A. Berlin, H. Engel, P. Recht, and J. Smeets (eds.). pp 319-332.

Anderson, E. V. 1978. Phasing Lead Out of Gasoline: Hard Knocks for Lead Alkyls Producers. Chem & Engr. News. February 6. pp 6-12.

Anonymous. 1975. Lead Contamination in Home Grown Vegetables. Chem. Eng. (Chementator), 82(11): 59.

Anonymous. 1975. High Levels of Lead in Boston Drinking Water Have Spurred Plans for a Plant. . . Chem. Eng. (Chementator), 82 (13):76.

Barltrop, D. 1973. Sources and Significance of Environmental Lead for Children.In: Proceedings, International Symposium, Environmental Health Aspects of Lead, Amsterdam, Oct. 2-6, 1972, D. Barth, A. Berlin, H. Engel, P. Recht, and J. Smeets (eds.) pp. 675-681.

Barltrop, D., and N. J. P. Killala. 1967. Faecal Excretion of Lead by Children, Lancet.2:1017-1019.

Bell, M. A., R. A. Ewing, G. A. Lutz, V. L. Holoman, B. Paris, and H. H. Krause. 1978. Reviews of the Environmental Effects of Pollutants. VII. Lead. EPA 600/1-78-029. U. S. Environmental Protection Agency, Health Effects Research Laboratory, Cincinnati, Ohio. Various pagination.

Bergner, K. G., and U. Rudt. 1968. Zur Bleiund Zinnaufnahme von Fetten und Fettreichen Lebeschmitteln in Wissblechdosen. (Absorption of Lead and Tin by Fats and Foods Rich in Fat in Tin Cans, Z. Lebensm. - Unters.-Forsch., 137(6):337-351 (Abstract only).

Bertinuson, J. R., and C. S. Clark. 1973. The Contribution to Lead Content of Soils from Urban Housing. Interface, 2 (1):6.

Bethea, R. M., and N. J. Bethea. 1975. Consequences of Lead in the Ambient Environment: An Analysis, Residue Reviews.54:55-77.

Bogden, J. D., and D. B. Louria. 1975. Soil Contamination from Lead in Paint Chips. Bull. Environ. Contam. Toxicol. 14(3):289-294.

Brandt, M., and Bentz, J. M. 1971. A Cooperative Study of the Determination of Lead in Milk, Microchemical Journal, 16: 113-120.

Brown, H. G., C. P. Hensley, G. L. McKinney, and J. L. Robinson, 1973. Efficiency of Heavy Metals Removal in Municipal Sewage Treatment Plants. Environ. Letters, 5(2):103-114.

Chisolm, J. J. Jr., and H. E. Harrison. 1956. The Exposure of Children to Lead, Pediatrics.18:943-958.

Cholak, J. 1964. Further Investigations of Atmospheric Concentration of Lead. Arch. Environ. Health. 8(2):314-324.

Chow, T. J. 1968. Isotope Analysis of Seawater by Mass Spectrometry. J. Water Poll. Contr. Fed., 40(3, pt.1). 399-411.

Chow, T. J., J. L. Earl, and C. B. Snyder, 1972. Lead Aerosol Baseline: Concentration at White Mountain and Laguna Mountain, California. Science. 178(4059):401-402.

Chow, T. J. 1973. Our Daily Lead. Chemistry in Britain. 9(6):258-263.

Clark, K. G. A., 1972. Lead-Glazed Earthenware, Lancet, 2(7778): 662-663.

Cogbill, E. C. and M. E. Hobbs. 1957. Transfer of Metallic Constituents of Cigarettes to the Main Stream Smoke. Tobacco Science.1:68-73.

Colwell, D. M., and Hicksman, A. J. 1973. The Concentration of Volatile and Particulate Lead Compounds in the Atmosphere: Measurements at Four Road Sites, Transport and Road Research Laboratory Report LR 545, Crowthorne, Berkshire. 5 pp.

Council for Agricultural Science and Technology. 1976. Application of Sewage Sludge to Cropland: Appraisal of Potential Hazards of the Heavy Metals to Plants and Animals. CAST (Council for Agricultural Science and Technology) Report No. 64. November 22, 1976.

Council on Environmental Quality. 1976. Environmental Quality - 1976. 7th Annual Report. Washington, D. C. 269 pp.

Daines, R. H., Motto, Harry, and Chilko, D. M. 1970. Atmospheric Lead-Its Relationship to Traffic Volume and Proximity to Highways. Environ. Sci. and Technol. 4(4):318-322.

Daines, R. H., D. W. Smith, A. F. Feliciano, and J. R. Trout. 1972. Air Levels of Lead Inside and Outside Homes. Industrial Medicine and Surgery. 41(10):26-28.

Dalton, E. F., and A. J. Malanoski. 1969. Atomic Absorption Analysis of Copper and Lead in Meat and Meat Products. J. Assoc. Official Anal. Chemists, 52(5): 1035-1038.

Davies, P. H., and W. H. Everhart. 1973. Effects of Chemical Variations in Aquatic Environments. Vol. III. Lead Toxicity to Rainbow Trout and Testing Application Factor Concept, Office of Research and Monitoring, U.S. Environmental Protection Agency, Washington, D.C. EPA-R3-73-011c, 80 pp.

Davis, R. B. 1977. Personal communication with John Litchfield, Battelle-Columbus Laboratories. Memo dated April 5, 1977, Reese B. Davis, Assistant Director for Packaging Technology, Bureau of Foods, Food and Drug Administration, Department of Health, Education and Welfare. Washington, D. C.

Day, J. P., M. Hart, and M. S. Robinson. 1975. Lead in Urban Street Dust. Nature.253:343-345.

Dedolph, R. G., Ter Haar, R. Holtzman, and H. Lucas, Jr. 1970. Sources of Lead in Perennial Ryegrass and Radishes, Environ. Sci. Technol. 4:217-223.

de Treville, R. T. P. 1964. Natural Occurrence of Lead, Arch. of Environ. Health. 8(2): 212-221.

Dorn, C. R., J. O. Pierce, II, G. R. Chase and P. E. Phillips. 1972. Lead, Copper, Zinc and Cadmium Contamination in Food Chains of Man, U.S. National Technical Information Service, Springfield, Va., PB-223 018/2, pp 121.

E. I. du Pont de Nemours and Co., Inc. 1978. Fall 1978 ESCON Forecast. Tech. Brief No. 7812. 5pp + Att., Wilmington, Delaware. September 22.

Durum, W. H. 1974. Occurrence of Some Trace Metals in Surface Waters and Groundwaters. In: Proceedings Sixteenth Water Quality Conference. Trace Metals in Water Supplies: Occurrence Significance and Control February 12-13, 1974. Champaign, Illinois. University of Illinois. pp 17-25.

Durum, W. H., J. D. Hern, and S. G. Heidel. 1971. Reconnaissance of Selected Minor Elements in Surface Waters of the U. S., U. S. Department of Interior, Washington, D. C., Geologic Survey Circular No. 643. 49 pp.

Durum, W. H., and J. D. Hem. 1972. An Overview of Trace Element Distribution Patterns in Water. In: Geochemical Environment in Relation to Health and Disease, Ann. N.Y. Acad. Sci., Vol. 199, H. C. Hopps, and H. L. Cannon (eds.), pp 26-36.

Dutilh, C. E., and Das, H. A. 1971. The Determination of Lead in Milk Powder, Radiochem. Radioannal. Letters. 6(3): 195-201.

Engel, R. E., E. I. Hammer, R. J. M. Horton, N. M. Lane, and L. A. Plumlee. 1971. Environmental lead and Public Health, Air Pollution Control Office, U.S. Environmental Protection Agency, Research Triangle Park, N. C., Publication No. AP-90, pp 34.

Engst, R., and H. Waggon. 1965. Uptake of Heavy Metals Traces in Canned Evaporated Milk, Nahrung 9: 859. (Ger.).

Environmental Health Resource Center. 1973. Health Effects and Recommendations for Atmospheric Lead, Cadmium, Mercury, and Asbestos. Illinois Institute for Environmental Quality, Chicago, Illinois. 101 pp.

Ewing, B. B., and J. E. Pearson. 1974. Lead in the Environment. From Advances in Environmental Science and Technology, Vol. 3. J. N. Pitts, Jr., and K. L. Metcalf, Editors. 126 pp.

Faoro, R. B. and T. B. McMullen. 1977. National Trends in Trace Metals in Ambient Air, EPA-450/1-77-003, U. S. Environmental Protection Agency, Research Triangle Park, North Carolina, Office of Air Quality Planning and Standards. 33 pp.

Fomon, S. J. 1974. Infant Nutrition, 2nd Ed., W. B. Saunders and Co., pp 8-9.

Guss, S. B. 1970. Contamination of Dairy Foods by Air Pollution, J. of Milk and Food Technol. 33(12): 553; 561; 567.

Haley, T. J. 1969. A Review of the Toxicology of Lead. Air Quality Monograph, No. 69-7. American Petroleum Institute, New York, New York.

Hammond, P. B. 1978. General Summary/Environmental Assessment. In: Review of The Environmental Effects of Pollutants. VII. Lead. EPA 600/1-78-029. U. S. Environmental Protection Agency, Health Effects Research Laboratory, Cincinnati, Ohio. July. pp 1.1-1.8.

Hankin, L., G. H. Heichel and R. A. Botsford. 1975. Lead in Pet Foods and Processed Organ Meats, A Human Problem? J. Amer. Med. Assn. 231(5): 484-485.

Harley, J. H. 1970. Discussion of Sources of Lead in Perennial Ryegrass and Radishes. Environ. Sci. Technol. 4: 225.

Harris, R. W., and W. R. Elsea, 1967. Ceramic Glaze as a Source of Lead Poisoning, J. Am. Med. Assoc., 202: 544-546.

Harrison, R. M., R. Perry, and D. H. Slater. 1975. The Contribution of Organic Lead Compounds to Total Lead Levels in Urban Atmospheres. In: International Symposium Proceedings, Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Vol. III. Luxembourg, Comm. of European Communities, pp. 1783-1788.

Hemphill, D. D., C. J. Marienfeld, R. S. Rerula, and J. O. Pierce. 1974. Roadside Lead Contamination in the Missouri Lead Belt. Arch Environ. Health, 28(4):190-194.

Holliday, M. A., A. S. Anderson, L. A. Barness, G. B. Forbes, R. B. Goodbloom, J. C. Haworth, A. M. Mauer, C. R. Scriver, W. B. Well, Jr., and M. Winick. 1974. Lead Contamination in Milks fed to Infants: 1972-1973. Review by the Committee on Nutrition, Pediatrics.53(52): 146.

Hunt, W. F. Jr., C. Pinkerton, O. McNulty, and J. Creason. 1971. A study in Trace Element Pollution of Air in 77 Midwestern Cities, In: Trace Substances in Environmental Health. IV, Proc. 4th annual conf., Trace Substances in Environ. Health, Univ. of Missouri, Columbia, Mo., D. D. Hemphill (ed.) pp. 56-68.

ILZRO, International Lead and Zinc Research Organization, Inc. 1972. Lead Research Digest No. 29, New York, New York. pp. 53.

Jacobs Engineering Co. 1976. Assessment of Hazardous Wast Practices in the Petroleum Refining Industry. Draft Report to U. S. Environmental Protection Agency.

Karalekas, P. C., G. F. Craun, A. F. Hammonds, C. R. Ryan, and D. J. Worth. 1975. Lead and Other Trace Metals in Drinking Water in the Boston Metropolitan Area. Presented at the annual conference of the American Water Works Association, Minneapolis, Minnesota, June 8-13, 14 pp.

Karalekas, P. C., C. R. Ryan, C. D. Larson and F. B. Taylor. 1977. Alternative Methods for Controlling The Corrosion of Lead Pipe. Presented at The Annual Conference of The New England Water Works Association, September 13.

Kehoe, R. A. 1961. The Metabolism of Lead in Health and Disease: The Harben Lectures, Journal of the Royal Institute of Public Health and Hygiene. 24: 1-81, 101-120, 129-143, 177-203.

Kehoe, R. A. 1976. Pharmacology and Toxicology of Heavy Metals: Lead Pharmacology and Therapeutics 1(2): 161-188.

Kehoe, R. A., J. R. Goldsmith and A. C. Hexter. 1968. Lead Intake From Food and From the Atmosphere. Science.149(3818): 1000.

Kehoe, R. A., F. Thamann and J. Cholak. 1933. On the Normal Absorption and Excretion of Lead. I. Lead Absorption and Excretion in Primitive Life. J. Indust. Hygiene, 15:320.

Khan, M. A. Q., W. F. Coello, and Z. A. Saleem. 1973. Lead Content of Soils Along Chicago's Eisenhower and Loop-Terminal Expressways. Arch. Environ. Contam. Toxicol., 1(3):209-223.

King, B. G. 1971. Maximum Daily Permissible Intake of Lead Without Excessive Body Burden in Children. American J. of Diseases of Childhood.122: 337.

Kirkpatrick, D. C., and D. C. Coffin. 1973. Cadmium, Mercury, and Lead Content of Various Cured Meats. J. Fd. Sci. Agr. 24(12): 1595-1598.

Klein, D. H. 1972. Mercury and Other Metals in Urban Soils. Environ. Sci. Technol. 6(6):560-562.

Klein, M., and R. Namer, 1970. Lead Release from Earthenware, N. Engl. J. Med. 283(23): 1292.

Kolbye, A. C. J., K. R. Mahaffey, J. A. Fiorino, P. C. Corneliussen and C. F. Jelinek. 1974. Food Exposures to Lead. Environ. Health Perspect. 7: 65-74.

Lagerwerff, J. V., and A. W. Specht. 1970. Contamination of Roadside Soil and Vegetation with Cadmium, Nichel, Lead, and Zinc. Environ. Sci. Technol. 4(7):583-586.

Lamm, S. H., Cole, B. L., Glynn K. L., and Ullman, W. W. 1973. Lead Content of Milks Fed to Infants - 1971-1972, New England Journal of Medicine, 289: 574.

Lamm, S. H., and J. F. Rosen. 1974. Lead Contamination of Milks Fed to Infants: 1972-1973. Pediatrics.54(2): 137-141.

Landrigan, P. J., S. H. Gehlbach, B. F. Rosenblum, J. M. Shoults, R. M. Candelaria, W. F. Barthel, J. A. Liddle, A. L. Smrek, M. W. Staehling, and J. F. Sanders. 1975. Epidemic Lead Absorption Near an Ore Smelter. New England J. of Med. 292,3,123-129.

Laveskog, A. 1970. A Method for Determining the Tetramethyl Lead (TML) and Tetraethyl Lead (TEL) in Air. Presented to the Second International Clean Air Congress of the International Union of Air Pollution Prevention Associations. Washington, D. C., December 6-11.

Lazrus, A. L., Lorange, Elizabeth, and Lodge, J. P., Jr. 1970. Lead and Other Metal Ions in United States Precipitation. Environ. Sci. Technol. 4(1):55-58.

Lanzola, E., M. Allegrini, and F. Breuer. 1973. Lead Levels in Infant Food Sold on the Italian Market. In: Proceedings of the International Symposium on Environmental Health Aspects of Lead Amsterdam, Oct. 2-6, 1972, D. Barth, A. Berlin, R. Engel, P. Recht and J. Smeets (eds.) pp 333-343.

Leland, H. V., E. D. Coperhaver and D. J. Wilkes. 1975. Heavy Metals and Other Trace Elements. J. Water Poll. Control Assoc. 47(6): 1635-1656.

Lepow, M. L., L. Bruckman, M. Gillette, S. Markowitz, R. Robino, and J. Kapish. 1975. Investigations into Sources of Lead in the Environment of Urban Children. Env. Research. 10:415-426.

Lewis, K. H. 1966. The Diet as a Source of Lead Pollution. In: Symposium on Environmental Lead Contamination, Sponsored by the United States Public Health Service, Dec. 13-15, 1965, Public Health Service Publication 1440, Washington, D. C., pp 17-20.

Lin-Fu, J. S. 1972. Undue Absorption of Lead Among Children - A New Look at an Old Problem. New England J. of Med. 286(13): 702-710.

Lautit, M. W., F. M. Patrick and R. S. Mathus. 1973. The Role of Metal - Concentrating Bacteria in a Food Chain in a River Receiving Effluent. University Otago. Med. School. 51(2): 37-38.

Lynch, G. P., D. G. Cornell and D. F. Smith. 1974. Excretion of Cadmium and Lead into Milk. In: Trace Element Metabolism in Animals - 2, W. G. Hoekstra, J. W. Suttie, H. E. Ganther and W. Mertz (eds.), University Park Press, Baltimore, pp 470-472.

Mahaffey, K. R., P. E. Corneliussen, C. F. Jelinek, and J. A. Fiorino. 1975. Heavy Metal Exposures From Foods, Environ. Health Perspect. 12: 63-69.

Mahaffey, K. R. 1978. Environmental Exposure to Lead. In: The Biogeochemistry of Lead in the Environment, Part B. J. O. Nriagu, (ed.). Elsevier/ North Holland Biomedical Press, Amsterdam. pp 1-36.

Martinez, J. 1977. Ross Laboratories Division of Abbott Labs, Columbus, Ohio. Personal Communication.

McCabe, L. J. and J. C. Vaughn. 1969. Trace Metals Content of Drinking Water from a Large System. Natl. Meeting, American Chemical Society, Minneapolis.

McCabe, L. J., J. M. Symons, R. D. Lee, and G. G. Robeck. 1970. Survey of Community Water Systems. J. Am. Water Works Assoc. 62(11):670-687.

McMullen, T. B., and R. B. Faoro. 1976. Comparing The Occurrences of Metals in Airborne Particulates and in Surficial Materials.Unpublished Internal Report, U. S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, 15 pp.

Mehani, S., 1966. Lead Retention by the Lungs of Lead-Exposed Workers. Ann. Occup. Hyg. 9(3): 165-171.

Meranger, J. C. 1970. The Heavy Metal Content of Fruit Juices and Carbonated Beverages by Atomic Absorption Spectrophotometry. Bull. Environ. Contam. Toxicol. 5(3): 271-275.

Ministry of Agriculture, Fisheries and Food. 1975. Working Party on the Monitoring of Foodstuffs for Heavy Metals, Fifth Report: Survey of Lead in Food: First Supplementary Report. Ministry of Agriculture, Fisheries and Food. Her Majesty's Stationery Office, London.

Mitchell, D. G. and K. M. Aldous. 1974. Lead Content of Foodstuffs, Environ. Health Perspect. 7: 59-64.

Motto, H. L., R. H. Daines, D. M. Chilko, and C. K. Motto. 1970. Lead in Soils and Plants: Its Relationship to Traffic Volume and Provimity to Highways. Environ. Sci. Technol. 4(3): 231-238.

Murthy, G. K., and U. S. Rhea. 1971. Cadmium, Copper, Iron, Lead, Manganese and Zinc in Evaporated Milk, Infant Products and Human Milk. J. of Dairy Sci. 54(7): 1001-1005.

Murthy, G. K., U. Rhea, and J. T. Peeler. 1967. Rubidium and Lead Content of Market Milk. J. Dairy Sci. 54(7): 1001-1005.

Murthy, G. K., U. Rhea and J. T. Peeler. 1971. Levels of Antimony, Cadmium, Chromium, Cobalt, Manganese and Zinc in Institutional Total Diets. Environ. Sci. Technol. 5: 436-442.

National Academy of Sciences, Committee on Biologic Effects of Atmospheric Pollutants. 1972. Biologic Effects of Atmospheric Pollutants. Lead. Airborne Lead in Perspective, Washington, D. C. pp 341.

National Academy of Sciences. 1976. Recommendations for the Prevention of Lead Poisoning in Children. Committee on Toxicology, Washington, D.C., July,65pp.

National Food Processors Association. 1977. (Formerly National Canners Association). CMI-NCA Lead in Canned Food Survey, and CMI-NCA Lead in Raw Product Survey, Washington, D. C.

Natusch, D. D. S., and J. R. Wallace. 1974. Urban Aerosal Toxicity: The Influence of Particle Size, Science. 186(4165):695-699. November 22.

Nozaki, K. 1966. Method for studies on Inhaled Particles in Human Respiratory System and Retention of Lead Fume, Industrial Health (Japan) 4 (X): 118-128.

Olson, K. W., and R. K. Skogerboe. 1975. Identification of Soil Lead Compounds from Automotive Sources, Environ. Sci. Technol. 9(3):227-230.

Page, A. L., and T. J. Ganje. 1970. Accumulations of Lead in Soils for Regions of High and Low Motor Vehicle Traffic Density, Environ. Sci. Technol. 4(2):140-142.

Palmisano, P. A., R. C. Sneed, and G. Cassady. 1969. Untaxed Whiskey and Fatal Lead Exposure. J. Pediat. 75(5): 869-872.

Patterson, C. C. 1965. Contaminated and Natural Lead Environments of Man, Arch. Environ. Health, 11: 344-363.

Patterson, C. C. 1978. California Institute of Technology, Division of Geological and Planetary Sciences. Personal Communication to R. A. Ewing.

Patterson, J. W., P. Shimada, and C. N. Haas. 1975. Heavy Metals Transport Through Municipal Sewage Treatment Plants. Proc. 2nd Natl. Conf. on Complete Water Reuse. Chicago, Ill., May 4-8.

Pickering, R. J. 1976. Summary of Toxic Substances in Water and Stream Sediments, Memorandum Report to Committee on Merchant Marine and Fisheries, U. S. House of Representatives, U. S. Geological Survey. Washington, D. C., 14 pp.

Pierrard, J. M. 1969. Photochemical Decomposition of Lead Halides from Automobile Exhaust. Environ. Sci. Technol. 3(1):48-51

Pinkerton, C., and D. I. Hammer, K. Bridbord, J. P. Creason, J. L. Kent, and G. K. Murthy. 1973. Human Milk as a Dietary Source of Cadmium and Lead, in: Trace Substances in Environmental Health - VI. Proceedings of the 6th Annual Conference on Trace Substances in Environmental Health. University of Missouri, Columbia, Missouri, June 13-15, 1972, D. D. Hemphill (ed.), pp 39-43.

Proctor, P. D., G. Kisvarsanyi, E. Garrison, and A. Williams. 1973. Heavy Metal Content of Surface and Groundwaters of the Springfield-Joplin Areas, Missouri, In: Trace Susbtances in environmental Health - VII, Proc. 7th Ann. Conf. Trace Substances in Environ. Health, University of Missouri, Columbia, Mo., June 12-14. D. D. Hemphill (ed.), pp 63-73.

Purdue, L. J., R. E. Erione, R. J. Thompson, and B. A. Bonfill. 1973. Determination of Organic and Total Lead in the Atmosphere by Atomic Absorption Spectrometry. Anal. Chem. 45:527-530.

Rathjen, J. 1978. U. S. Bureau of Mines. Personal Communication to R. A. Ewing.

Rhodes, R. C. and D. H.Fair. 1978. Air quality Data for Metals, 1975, from the National Air Surveillance Networks, EPA-600/4-78-059, U. S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory, Research Triangle Park, North Carolina. October. 104 pp.

Roberts, H. R. 1978. Statement before The EPA Public Hearing on Proposed National Ambient Air Quality Standard for Lead. Washington, D. C. February 15.

Roberts, T. M., T. C. Hutchinson, J. Paciga, A. Chattopadbyay, R. E. Van Loon, and D. K. Parkinson. 1974. Lead Contamination Around Secondary Smelters. Estimates of Dispersal and Accumulation by Humans. Science, 186(4169):1120-1123.

Robinson, E., and F. Ludwig. 1967. Size Distributions of Atmospheric Lead Aerosols. J. Air Poll. Control Assoc. 17: 664-669.

Ryan, C. R. 1978. Region I, U. S. Environmental Protection Agency. Personal communication to R. A. Ewing.

Sarett, H. D. 1974. Lead Contamination in Milks Fed to Infants: 1972-1973 Comments. Pediatrics, 53(2): 143-144.

Sartor, J. D. and G. B. Boyd. 1972. Water Pollution Aspects of Street Surface Contaminants, U. S. Environmental Protection Agency, EPA-R2-72-081. Washington, D. C.

Schaule, D., and C. C. Patterson. 1978. The Occurrence of Lead in The Northeast Pacific and The Effects of Anthropogenic Inputs. To be published in: Proceedings of an International Experts Discussion on Lead: Occurrence, Fate, and Pollution in The Marine Environment, Rovinj, Yugoslavia, October, 1977. M. Branica (ed.) Pergamon Press, Oxford, England.

Schroeder, H. A., J. J. Balassa, F. S. Gibson and S. N. Valanju. 1961. Abnormal Trace Metals in Man: Lead, J. Chron. Dis. 14(4): 408-425.

Schubert, J., A. Brodsky, and S. Tyler. 1967. The Log-normal Function as a Stochastic Model of the Distribution of Strontium - 90 and Other Fission Products in Humans. Health Physics. 13: 1187-1204.

Schuck, E. A., 1970. Discussion: Lead in Soils and Plants: Its Relationship to Traffic Volume and Proximity to Highways, Environ. Sci. Technol. 4(3): 238.

Schuck, E. A., and J. K. Locke, 1970. Relationship of Automotive Lead Particulates to Certain Consumer Crops, Environ. Sci. Technol. 4(4): 324-330.

Shacklette, H. T., J. C. Hamilton, J. G. Boerngen, and J. M. Bowles. 1971. Elemental Composition of Surficial Materials in the Conterminous United States, U. S. Geological Survey, Prof. Paper 574-0. 71 pp.

Solomon, R. L., and D. F. S. Natusch. 1977. Environmental Contamination by Lead and Other Heavy Metals. Vol III. Distribution and Characterization of Urban Dusts. Final Report, NSF RANN Grants. Institute for Environmental Studies, Urbana - Champaign, Illinois. 96 pp.

Tepper, L. B. and L. S. Levin. 1975. A Survey of Air and Population Lead Levels in Selected American Communities. In: Lead. T. B. Griffin and J. H. Knelson (eds.). Environmental Quality and Safety. Supplement Vol. II. Georg. Thieme Publishers, Stuttgart. pp 152-196.

Ter Haar, G., 1970. Air As A Source of Lead in Edible Crops, Environmental Science and Technology, 4(3): 226-229.

Ter Haar, G. L., and R. Aronow. 1974. New Information of Lead in Diet and Dust as Related to the Childhood Lead Problem, Environ. Health Perspect. Experimental Issue.(7):83-89.

Ter Haar, G. L., and M. A. Bayard. 1971. Composition of Airborne Lead Particles, Nature. 232(5312):553-554. August 20.

Thomas, B., J. A. Roghan, and E. D. Watters. 1972. Lead and Cadmium Content of Some Vegetable Foodstuffs. J. Sci. Food Agr., 23(12): 1493-1498.

Thomas, B., J. A. Roughan and E. D. Watters. 1973. Lead and Cadmium Content of Some Canned Fruit and Vegetables. J. of Science Agr. 24(3): 447-449.

Tidball, R. R. 1976. Lead in Soils. In: Lead in The Environment, U. S. Geological Survey Professional Paper 957. U. W. Department of the Interior, Washington, D. C. pp 45-52.

U. S. Bureau of Mines. 1978. Personal communication from J. Rathjen to R. A. Ewing.

U. S. Department of Agriculture. 1968a. Household Food Consumption Survey, Report No. 1, Food Consumption of Households in The United States, Spring, 1965. Agricultural Research Service, Washington, D. C. U. S. Government Printing Office.

U. S. Department of Agriculture. 1968b. Household Food Consumption Survey, Report No. 11. Food and Nutrient Intake of Individuals in The United States, Spring 1965. Agricultural Research Service, Washington, D. C. U. S. Governmnet Printing Office.

U. S. Department of Agriculture. 1974. Lead in Evaporated Milk and Evaporated Skim Milk, Proposed Tolerance. 39FR42740-42743.. December 6.

U. S. Department of Agriculture. 1978. Agricultural Statistics, 1977, Washington, D. C.

U. S. Department of Health, Education, and Welfare. 1965a. Survey of Lead in the Atmosphere of Three Urban Communities. Working Group of Lead Contamination, Division of Air Pollution, Public Health Service, Cincinnati, Ohio. PHS Publication No. 999-AP-12, pp 94.

U. S. Department of Health, Education, and Welfare. 1965b. Revision of Air Pollution Survey of Lead in the Atmosphere of Three Urban Communities. U. S. Public Health Service, Publication 999-AP-12, Cincinnati, Ohio, 94 pp.

U. S. Department of Health, Education and Welfare, Food and Drug Administration. 1975. Compliance Program Evaluation FY 1974 Heavy Metals in Foods Survey, Bureau of Foods, Report dated: June 19, 1975.

U.S. Department of Health, Education and Welfare. 1977. FDA Compliance Program Evaluation - FY 1974 Total Diet Studies, Public Health Service, Food and Drug Administration - Bureau of Foods, pp 16; 22 Tables.

U. S. Department of Labor. 1975. Lead-Occupational Exposure; Proposed Standard. Occupational Safety and Health Administration. 40FR45134-45948. October 3.

U. S. Department of Labor. 1978a. Occupational Exposure to Lead. Final Standard. Occupational Safety and Health Administration, Washington, D. C. 43 FR 52952-53014. November 14.

U. S. Department of Labor. 1978b. Occupational Exposure to Lead. Attachments to the Preamble for the Final Standard. Occupational Safety and Health Administration, Washington, D. C. 43 FR 54354 54509. November 21.

U. S. Environmental Protection Agency. 1973c. Development Document for Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Major Organic Products Segment of the Organic Chemicals Manufacturing Point Source Category, EPA 440/1-73/009, Washington, D. C. December.

U. S. Environmental Protection Agency. 1978b. National Primary and Secondary Air Quality Standard for Lead. 43 FR 46246-46263. October 5.

U. S. Environmental Protection Agency. 1978c. Implementation Plans for Lead National Ambient Air Quality Standard. 43 FR 46264-46271. October 5.

U. S. Environmental Protection Agency, Health Effects Branch Processes and Effects Division, Office of Research and Monitoring. 1972. EPA's Position on the Health Effects of Airborne Lead, Washington, D.C.

U. S. Environmental Protection Agency. 1975f. Chemical Analysis of Interstate Carrier Water Supply System, EPA 430/9-75-005. U. S. Environmental Protection Agency, Washington, D. C. 90 pp.

U. S. Environmental Protection Agency. 1975g. National Interim Primary Drinking Water Regulation. 40 CFR Part 141 Washington, D. C. 40 FR 1190-11998. March 14.

U. S. Environmental Protection Agency. 1976. Assessment of Industrial Hazardous Waste Practices - Organic Chemicals, Pesticides and Explosives Industries, EPA Report SW-118c, Washington, D. C.

U. S. Environmental Protection Agency. 1976c. Assessment of Industrial Hazardous Waste Practices: Paint and Allied Products Industry, Contract Solvent Reclaiming Operations, and Factory Application of Coatings, Report SW-119c.

U. S. Environmental Protection Agency. 1977. Air Quality Criteria for Lead. EPA 600/8-77-017. U. S. Environmental Protection Agency, office of Research and Development, Washington, D. C. December. Various pagination.

U. S. Environmental Protection Agency. 1978a. Title 40 - Protection of Environment. Effluent Limitations Guidelines for Existing Sources, Ore Mining and Dressing Point Source Category. Washington, D. C. 43 FR 29771-29782. July 11.

U. S. Environmental Protection Agency. 1978b. National Primary and Secondary Air Quality Standard for Lead. 43 FR 46264-46271.

Vandecaveye, S. C., G. M. Horner, and C. M. Keaton. 1936. Unproductiveness of Certain Orchard Soils as Related to Lead Arsenate Spray Accumulations. Soil Sci. 42:203-215.

Walls, A. D. F. 1969. Home Made Cider: Source of Lead Poisoning, British Med. J. 1: 98.

Warren, H. V. and R. E. Delavault. 1962. Lead in Some Food Crops and Trees, J. Sci. Fd. Agr. 13:96-98.

Wixson, B. G., E. Bolter, N. L. Gale, J. C. Jennett, and K. Purushothaman. 1972. The Lead Industry as a Source of Trace Metals in the Environment. Proc. Environmental Resources Conf. on Cycling and Control of Metals, Columbus, Ohio, Battelle Memorial Inst. pp 11.

Wong, C. S., and P. Berrang. 1976. Contamination of Tap Water by Lead Pipe and Solder. Bull. of Env. Contam. and Toxicol. 15(5) 530-534.

World Health Organization. 1967. Specifications for the Identity and Purity of Food Additives and their Toxicological Evaluation: Some Emulsifiers and Stabilizers and Certain Other Substances, World Health Organization Technical Report Series No. 373.

World Health Organization. 1972. Evaluation of Certain Food Additives. Joint FAO/WHO Committee on Food Additives. WHO Technical Report Series.

World Health Organization, 1977. Lead: Environmental Health Criteria 3. United Nations Environmental Program and The World Health Organization, Geneva. 160 pp.

Yankel, A. J., I. H. Von Lindern and S. D. Walter. 1977. The Silver Valley Lead Study: The Relationship Between Childhood Blood Lead Levels and Environmental Exposure. J. Air Poll. Control Assoc. 27(8):763-767.

Zook, E. G. J. J. Powell, B. M. Hackley, J. A. Emerson, J. R. Brooker, and G. M. Knobl, Jr. 1976. National Marine Fisheries Service Preliminary Survey of Selected Seafoods for Mercury, Lead Cadmium, Chromium and Arsenic Content, J. Agr. and Food Content, 24(1): 47-53.

9.0 ASSESSMENT OF THE NEED FOR AND POSSIBLE APPROACHES TO LIMITATIONS ON LEAD

9.1 SUMMARY

Lead is undeniably a toxic element, and lead poisoning has been a problem for mankind for centuries. Adverse effects of lead on health vary from acute lead poisoning, which is easily recognized and on which there is general agreement down to levels where observable effects are debatable and sometimes controversial. However, even low levels cannot be dismissed as inconsequential; lead and its compounds do not biodegrade to innocuous residues, but persist for an infinite time.

The evidence is overwhelming that there is a critical need for limitations which will prevent the occurrence of acute lead poisoning, and regulations have been enacted or proposed in this country which should minimize its future occurrence from presently acceptable uses and processes involving lead. Some occupational problems still remain, principally at primary and secondary lead smelters, and in lead-acid storage battery plants (see Section 7.5.3); other specialized employee groups susceptible to acute lead poisoning are painters, scrapyard workers, and shipbreakers.

This is primarily an occupational problem requiring specific control measures. Such measures, e.g., recent regulations reducing allowable work place air lead concentrations from 200 to 50 $\mu g/m^3$, have been promulgated by The Occupational Safety and Health Administration of The Department of Labor.

The major nonoccupational acute lead poisoning problem remaining is that of children, primarily as a result of exposure to high-lead paint and dust in old substandard housing.

Although recently-enacted limitations on lead-based paint will prevent today's paints from presenting future problems of this sort, no current limitations appear able to solve this existing problem, nor do limitations appear well suited to address this problem. Greatly expanded surveillance and pediatric management appear to offer the optimum approach to this lead poisoning problem.

The need for limitations that will protect the general public which is nonoccupationally exposed is less conclusive than the need to prevent clinical lead poisoning. Part of the uncertainty relates to the changing concentrations of environmental lead.

Historically, ambient concentrations and environmental exposure to lead have been increasing almost since the Industrial Revolution. Fortunately, the rate of increase in environmental concentrations appears to have stabilized somewhat in recent years. Actually, the trend towards ever-increasing concentrations may finally have been halted and reversed by some of the limitations already enacted, e.g. the phasedown of leaded gasolines, but the benefits of these are not yet fully realized, nor even totally predictable.

In spite of these impending improvements, exposure to lead cannot be demonstrated to be below the level at which no adverse effects occur. Part of the problem is that this level cannot be unequivocally defined, since the health significance of the subtle biochemical disturbances is subject to varying interpretations. Nevertheless, a case can be made on the basis that the mean blood lead levels of the nonoccupationally exposed general population, at 15-20 μg/dl, if not at the level where irreversible effects begin to occur, is so close to it that the margin of safety is insufficient. Prudence dictates that this margin be increased, if feasible, i.e. there is a need for additional limitations to further reduce average blood lead levels.

The need is even greater to reduce blood lead levels in children, who constitute an especially sensitive segment of the general population; the margin of safety between present levels of blood lead in children and tolerable levels is relatively small, possibly the smallest for any toxic substance to which children are exposed.

The optimum approach to a further reduction in exposure to lead was identified by the analysis of man's sources of lead intake (Section 8), which demonstrated the importance of ingested lead in determining blood lead levels, and also that food and drink were the major contributors. An analysis of other sources of lead exposure reinforced the importance of ingested lead and confirmed that this approach to limitations should receive high priority. This does not negate the importance of other limitations; however, a compilation of existing and proposed Federal limitations (Section 9.4) shows that most of the other pathways for lead exposure have already come under control.

Thus, control of adventitious lead in food and drink appeared to be the principal limitations yet to be instituted. As witnessed by the testimony presented at the EPA public hearing considering the proposed national ambient air quality standard on lead on February 15, 1978 by The Acting Director of the Bureau of Foods, Food and Drug Administration (Roberts, 1978), the FDA had concern for this problem and has an ongoing program designed for its maximum mitigation within the bounds of existing food can technology. This has been a cooperative program with the can manufacturing and food packing industries, with promising results, some of which are described herein.

At the time of the selection of adventitious lead in food as a high priority problem for consideration of possible limitations during the second phase of this program, it was not known that an FDA program was in progress in this area. The results of this program offer independent support to the approach towards this problem which was adopted by FDA, and it is hoped that they will be useful to the continuing FDA efforts.

In Section 9.5.2 alternatives to lead-soldered cans for the packaging of food are described and analyzed. These include cans with cemented and welded side seams, two-piece cans formed by drawing procedures, aluminum cans, glass containers and retortable pouches. The latter, a very recent development,are made of an aluminum-plastic laminate and have some significant advantages in protecting flavor during the sterilization process, as well as in eliminating lead. However, at this time the packing process is slow and therefore costly. As this process is developed and perfected it can be expected to become more competitive.

In contrast to the total replacement of the three-piece soldered can, the can industry has demonstrated over the last several years that a significant reduction in adventitious lead in canned foods is possible by a combination of evolutionary improvements in can making technology and "good manufacturing practices", including especially close attention to quality control. Illustrative of this is the 40+ percent decrease in lead content of 10 representative canned foods in a 1976 industry survey compared to the results of the similar survey conducted in 1974. The average lead content of the 10 foods surveyed was 0.19 ppm. Infant foods showed even larger reductions; the average lead content of 16 canned fruit juices surveyed in 1977 was 0.06 ppm, compared to the 1973 average of 0.29 ppm.

If these improvements can be extended industry-wide a major reduction in the average lead content of canned foods can be achieved rather quickly, and without the need for a large capital investment program by the industry.

Capital costs to adopt this approach of modifying present can making lines and cleanup of soldering operations are estimated by The Can Manufacturers Institute as moderate, of the order of $50,000 per line, or about $50 million for the industry. Adding equipment to apply an organic polymer stripe over the solder seam would increase the capital cost per line by approximately $20,000 or $20 million for the industry. It was estimated that added operating costs of $0.10 to $0.45 per thousand cans would be incurred.

The capital costs to entirely replace the three-piece soldered can with any of the lead-free forming processes were estimated to be in the $600,000 to $2,500.000 range per can line, resulting in an industry investment in the $1 to $3 billion dollar range. Added operating costs of perhaps $3 to 6 per thousand cans were projected.

The benefit-cost ratios of the alternatives strongly favor the can line modification approach now being implemented, and its adoption, at least as an interim improvement, has much to recommend it. With the further improvement of seam striping and/or sprayed organic polymer linings for infant foods, most of the benefits to be gained by reduction of adventitious lead in canned foods would be achieved.

Processed food is not the only possible source of ingested lead for the general population. Another potential, though minor source of ingested lead is from the lead glaze used on dinnerware. Lead glazes are the most widespread in use and are considered best for all ordinary purposes. If the glaze is improperly formulated and/or fired, the lead in the glaze is extractable by foods and beverages, and a few cases of clinical lead poisoning have occurred from this cause. This problem is one of individuals, with negligible effect on the general public. The U.S. Food and Drug Administration, which is responsible for the regulation of this "indirect food additive," monitors both domestic and foreign ware. Domestic dinnerware appears to have no problem in meeting lead release limitations; some foreign ware does. As of now, no fully suitable alternative to lead glazes appears to exist commercially.

Modern pewter contains no lead and hence poses a minimal lead ingestion problem; antique pewter does, but its use appears to be so limited as to be discounted. Almost no community drinking water systems exceed the primary lead drinking water standard of 50 ppb; in a few soft-water areas lead service piping to homes has caused high lead dontents in domestic water. This does not appear to be a widespread problem. Some concern has been expressed recently about the hazard from decal-decorated drinking glasses. Results obtained by the interagency task force formed to investigate this potential hazard led to the conclusion that no formal rule-making is required at this time.

9.2 NEED FOR LIMITATIONS

Lead is undeniably a toxic substance. The symptoms and effects of acute lead poisoning have been known for centuries. Clinical, epidemological, and toxicological studies have conclusively demonstrated that excessive exposure to lead adversely affects human health. These adverse effects vary from acute lead poisoning, easily recognized and on which there is general agreement, down through sub-clinical effects to more subtle biochemical disturbances of the heme-synthetic mechanism which occur at levels of lead exposure well below those that cause any observable decrement in hemoglobin (Hammond, 1977). Not only is this latter effect subtle but also variable, and not universally acknowledged.

9.2.1 Acute Lead Poisoning

A need for limitations may be categorized by whether it applies to the acute lead exposure case or to the low end of the scale. Considering first the upper end, there is very little disagreement, on either the adverse effects, the need to mitigate or eliminate the hazard, or the form of the limitations required to accomplish this. The need to prevent excessive exposure to lead, such as will induce acute lead poisoning, has long been recognized, as evidenced by the existence of numerous regulations designed to prevent or minimize such exposures, especially occupational exposures, both in the United States and abroad.

Although great strides have been made in the past half century in mitigating occupational lead exposure and industrial lead poisoning, the problem has not yet been entirely eliminated. Epidemiologic studies conducted by the Center for Disease Control in 1975 and 1976 in 5 different U.S. lead facilities have shown unacceptably high blood-lead levels and symptoms of lead poisoning in every plant studied (see Section 7.5.3.1). On the basis of the findings of this and other studies it is obvious that the currently accepted upper limit of a safe blood lead level (80 μg/dl) does not provide adequate protection against lead toxicity.

In the industrial situation the route of entry for lead is primarily inhalation rather than absorption or ingestion, and high blood lead levels can be correlated with high air lead concentrations. The data of Williams, cited in the OSHA proposed occupational exposure standards (U.S. Department of Labor, 1975), showed that an air lead concentration of 200 $\mu g/m^3$ corresponded to a mean blood lead level of 70 μg/dl (48-92 μg/dl range). Similarly, the Williams data suggest that air lead concentrations of 150 $\mu g/m^3$ correspond to a blood lead range of 38-82 μg/dl, with a mean blood lead level of 60 μg/dl.

Epidemiological studies are unequivocal in identifying a need for additional limitations on occupational exposure to lead to prevent overexposure and lead poisoning symptoms in lead industry workers. The Occupational Safety and Health Administration of the U.S. Department of Labor, which has the primary responsibility for safety in the workplace, has promulgated regulations to minimize or eliminate this risk by reducing the time weighted overage (8-hr day, 40-hr week) lead concentration in the workplace from 200 to 50 $\mu g/m^3$ (U.S. Department of Labor, 1978). The regulations also provide for, among other things, the monitoring of employee exposure, methods of compliance, housekeeping practices, personal protective equipment and clothing, training, medical surveillance, and record keeping.

On the basis of the data submitted in support of this regulation, it is concluded that it will indeed produce the desired results, and there is no demonstrated need at this time for additional limitations on occupational exposure to lead.

The major nonoccupational acute lead poisoning problem remaining, and unfortunately, one striking at the most susceptible segment of the population, children and infants, is that arising from ingestion of the high-lead interior and exterior paints (and dusts) generally associated with substandard urban housing. It has been estimated that several hundred thousand children in the United States, primarily from areas where such substandard housing is prevalent, may have blood leads in excess of 40 μg/dl (see Section 7.4). Possible approaches to this problem are discussed in Section 9.3.3.

While it is true that there have been a few isolated cases of acute lead poisoning in the U.S. from illicit whiskey, from improperly glazed ceramic beverage containers, from misuse of other lead products, and even from the excessive comsumption of a calcium diet supplement derived from animal bones, the frequency of such cases is small as compared to other sources of lead exposure.

9.2.2 Limitations at Low Lead Levels

As indicated by the previous discussion, there is widespread agreement on the characterization and the intrepretation of the effects of lead upon humans with acute or chronic lead poisoning. As lead dose becomes smaller and smaller, it's effects are less and less definitive and unequivocal, and at just-perceptible levels there is considerable uncertainty and disagreement upon its effects. Although it has long been assumed that low levels of lead can be tolerated in humans without demonstrable adverse effects, the exact levels of exposure that define these thresholds have not been clearly established.

It is evident that there is scientific controversy about facts and intrepretation of the health significance of subtle biochemical disturbances associated with blood levels below traditional levels of concern.

In this context, clinical effects can be considered to be those which a subject can perceive and which a physician would normally seek to correct. Subclinical effects are those which under normal circumstances do not result in a perceptible decrement in body function but which could possibly reduce an individual's capacity to cope with a coexistent body stress (Hammond, 1978).

Hammond (1978), reviewing the effects of lead upon man, noted that the principal organs affected by lead are the kidneys, the hematopoietic system, and the central and peripheral nervous systems, and that in all four cases a spectrum is seen, including both clinical and subclinical effects.

As Hammond (1978) points out:

> "The effects of lead on the hematopoietic system are varied in nature. They include shortened survival of circulating red cells and decreased rate of hemoglobin synthesis in the bone marrow. Both of these general effects contribute to the anemia which is characteristic of lead poisoning. Anemia is clearly a clinical effect, one which a physician would normally seek to correct. The minimal level of lead exposure which is necessary to cause anemia is difficult to specify, if only because the clinical definition of anemia is imprecise. It is perhaps more to the point to consider the minimal level of lead exposure at which a downward drift in blood hemoglobin first appears, even within the accepted normal range. This minimal level appears to be PbB = 40 in children and PbB = 50 in adults.
>
> Two enzymes in the pathway leading to heme synthesis are clearly inhibited by lead at PbB's below 40. The first of these is heme synthetase, a mitochondrial enzyme which catalyzes the final step in heme synthesis, the insertion of iron into protoporphyrin IX. Inhibition of this enzyme is reflected on the appearance of elevated protoporphyrin IX in circulating red cells, an effect which occurs at PbB's as low as 25 in adult males and as low as 20 in adult females and children. At even lower levels of lead exposure (PbB = 15), the enzyme aminolevulinic acid dehydratase (ALAD) which converts the the heme precursor aminolevulinic acid (ALA) to the intermediate porphobilinogen (PBG) is somewhat inhibited, both in blood and in some other tissues. The consequence of inhibition of this enzyme is thought to be elevation of the precursor ALA in blood plasma and urine. This effect does occur, but only as PbB rises above 40.

> Thus, the significance of ALAD inhibition at very low levels of lead exposure is uncertain and is considered by most to be a subcritical effect.
>
> The basic issue, for which there is no clear answer, is as to whether elevation of erythrocytic protoporphyrin between PbB = 20 and PbB = 40 is to be considered a critical or subcritical effect. The effect certainly is compensated since hemoglobin levels are sustained at a normal level in this range of lead exposure. The real issue is as to whether or not a person's capacity to cope with a co-existent stress on hematopoiesis is in any way compromised. Not much is known about this problem. There is a need for more research to determine the health consequences of this compensated state, and also to determine the implications of the effects of lead on the synthesis of other hemoproteins essential to normal body function."

It is well-known that very high lead exposure can cause severe, often fatal damage to the brain in both adults and children. Effects at lower exposures become increasingly difficult to define, and there has been much controversy in recent years regarding the effect of lead on the central nervous system. A number of recent studies examining possible neurological and behavioral effects of lead in children have been reported. Nearly all of these studies have been retrospective in nature, and have generally focused on children with blood leads above threshold levels (i.e.>25 μg/dl) for detectable changes in the hematopoietic system. Also, the experimental designs of these studies are flawed in one way or another, and the effects observed may have been a result of the combined effects of lead and other stressors, both physical and social, since these studies have almost invariably been conducted in socio-economically deprived populations.

Thus, while there may be threshold levels of lead exposure for subtle neurological and behavioral effects, it does not seem possible at this time to correlate these unequivocally with specific levels of either blood lead or lead intake.

Conceding that an unambiguous correspondence of cause and effect cannot be established at threshold levels of lead exposure, possibly the question of the most desirable course of action can be approached in another way.

As noted earlier, there is considerable scientific controversy about the intrepretation of the health effects of lead, and about the level at which irreversible effects come into play. In the Air Quality Criteria for Lead document (U.S. Enviromental Protection Agency, 1977) a tabulation of blood lead levels versus lowest - observed - effects levels was presented (Table 9.1).

TABLE 9.1 BLOOD LEAD LEVELS VERSUS LOWEST-OBSERVED-EFFECTS LEVELS

Lowest level for observed effects, μg Pb/dl whole blood	Observed effect	Population group
10	ALAD inhibition	Children and adults
15 to 20	Free erythrocyte porphyrin elevation	Adult females and children
20 to 25	Free erythrocyte porphyrin elevation	Adult males
40	Increased urinary ALA excretion	Children and adults
40	Anemia	Children
40	Coproporphyrin elevation	Adults and children
50	Anemia	Adults
50 to 60	Cognitive (CNS) deficits	Children
50 to 60	Peripheral neuropathies	Adults and children
80 to 100	Encephalopathy symptoms	Children
100 to 120	Encephalopathy symptoms	Adults

It was noted that the question of the inhibitory effect of lead on the enzyme delta-ALAD was overshadowed by the larger issue of the health significance of this observation, but that a value of 10 μg/dl appears to represent a threshold level. It was also noted that elevation of erythrocyte protoporphyrin is better accepted as an indicator of physiological impairment. On the basis of a number of studies, threshold values of blood lead at which erythrocyte protoporphyrin is elevated appear to be 15 to 20 μg/dl for children and adult females and 20 to 25 μg/dl for adult males.

Next, the question might be asked, how does this compare with average blood lead levels in the general population? This question really cannot be answered at this time; There are no data on such averages, for either adults or children, or even for large special segments of the population, e.g., the armed services. There have been numerous studies of blood lead levels in selected population segments, but these have been performed because of suspected or perceived problems, consequently the data are skewed towards high values. The fragmentary evidence available points toward a U.S. average (for adults) in the 15-20 μg/dl range. An extensive summary of blood lead levels reported from numerous investigations is contained in the Air Quality Criteria for Lead document, but the diversity of the results makes the derivation of an average difficult. For example, geometric means were calculated for the 11 geographic populations samples studied by Tepper and Levin (1975); These ranged from 12.5 to 20.6 μg/dl. All but three were above 15 μg/dl (see Table 8.37).

Even fewer data are available on children, especially "normal" children unexposed to above-average lead intake. Characteristically in the studies cited by the U.S. Environmental Protection Agency young (1977a) children had higher blood-lead values than older children (ages not specified) and adults in the same environment. Fine, et al., (1972) determined a mean blood lead value of 25.5 μg/dl for 6151 children in 14 intermediate sized cities in Illinois. As an approximation, it can be assumed that the average blood lead for children is in the 20-25 μg/dl range.

This lack of data hampers the evaluation of where the U.S. population stands now with respect to blood lead, and also what effects various alternative limitations might have. Nevertheless, the above approximations of blood lead levels will suffice for development of the hypothesis that additional limitations on lead intake are desirable for the general health. As noted in preceding paragraphs elevated protoporphyrin can occur at blood lead levels as low as 25 μg/dl in adult males and as low as 20 μg/dl in adult females and in children (Hammond,1978). There is continuing debate as to whether this is a critical or subcritical effect. However, the disquieting fact is that these effects extend down to potential threshold blood levels approaching the general U.S. average; and if there is no overlap, neither is there any distance between the two. Thus, even though the conclusion be accepted that threshold concentrations (and effects) of blood lead are higher than the elevation in erythrocyte protoporphyrin suggests, the fact remains that the margin of safety is small, and prudence suggests that it be increased.

The concern here is for the general population, and for the most susceptible segments of it, children and pregnant women. EPA has stated that the agency believes that young children (ages 1-5 years) should be regarded as the foremost critically sensitive population subgroup for setting of lead standards (U.S. Environmental Protection Agency, 1978). This is because hematologic and neurologic effects in children are shown to occur at lower thresholds than adults, and because children have a greater risk of exposure to non-food material containing lead, such as dust and soil (and old lead paint), as the result of normal hand-to-mouth activity. Additionally, as was noted in the supporting EPA Criteria Document (U.S. Environmental Protection Agency, 1977a) other factors suggesting that children may be at greater risk include:

(1) greater intake of lead via inhalation and ingestion per unit body weight;
(2) greater absorption and retention of ingested lead;
(3) physiologic stresses due to rapid growth rate and dietary habits;
(4) incomplete development of metabolic defense mechanisms;
(5) greater sensitivity of developing systems.

Pregnant women and the fetus are at risk because of transplacental movement of lead to the fetus and the possibility of maternal complications at delivery. Because there is a balance between maternal blood lead levels and fetal blood lead levels, concern exists that development of the nervous system of the fetus may be impaired due to neurotoxicity of lead. Changes in fetal heme synthesis and premature births have been associated with prenatal exposure of the fetus to lead. However, available evidence does not indicate that pregnant women and the fetus would require a more stringent standard than young children. (U.S. Environmental Protection Agency, 1978).

The very small fraction of the total population occupationally exposed to excessive levels of lead is a separate problem, requiring different measures.

The question of whether there is a need for limitations on lead has already been answered in the affirmative with respect to this group. Numerous limitations have already been enacted in the United States and around the world in response to this need.

With respect to the need for additional (new) limitations for the protection of the general population, one way to approach the analysis is by posing a series of questions:

(1) Is a need indicated for reductions in blood leads and body burdens?

As discussed above, there is believed to be an insufficient margin of safety between present blood lead levels and potentially undesirable ones. This margin of safety needs to be increased.

(2) What is the size of the population affected or at risk?

If it is accepted that average blood lead levels, in the 20 ± 5 μg/dl range, are at or close to levels where adverse effects, even through subtle, are possible, then the entire U.S. population (250+ million) may be affected or benefited, if the limitation is one which applies universally.

(3) Is any sensitive segment of the general population affected, or exposed to specific risk?

It has been shown and generally accepted that children, especially young children, are the most sensitive group, for a variety of reasons: frequent pica, speed of growth, susceptibility of the central nervous system, among others. They are closely followed by pregnant women: and women in the prime childbearing years of 20-34 should be protected against intake of too much lead, i.e. their blood lead should be maintained below 25 μg/dl.

(4) What is the prognosis for reductions in exposure to lead, in the absence of new limitations?

There will definitely be reductions in the exposure of the general population to lead from limitations already enacted, notably the phasedown in leaded gasoline use. As discussed in Section 8.2.3, this should reduce the national annual ambient air lead concentration from the 1975 mean of 0.7-0.8 $\mu g/m^3$ to below 0.3 $\mu g/m^3$ by 1980 or shortly thereafter. On the basis that 1 $\mu g/m^3$ in air lead in this concentration range is equivalent to approximately 1 to 2 μg/dl blood lead, this may reduce mean blood lead by about 0.5 to 1.0 μg/dl.

The new ambient air lead standard of 1.5 $\mu g/m^3$, will produce some reductions in lead exposure for the small segment of the population which is in the intermediate exposure category because of their location in the proximity of a major air emission source (e.g. smelter).

The general population, on the other hand, is expected to receive little additional benefit from an ambient air lead standard at 1.5 $\mu g/m^3$ in those locations where the combustion of leaded gasoline accounts for a high percentage of atmospheric lead. On the basis of EPA data from the National Air Surveillance Networks (NASN) there were very few stations which had annual or quarterly means exceeding this level in 1975 (Section 8.2.1). The phasedown may already have taken these areas below the 1.5 $\mu g/m^3$ level, or will very soon. Accordingly, this ambient air standard will do little or nothing to reduce the air lead intake of the general population.

Extension of effluent guidelines and promulgation of pretreatment standards will contribute to reducing lead concentrations in industrial wastewaters in special areas where it is now high. Since half or more of the lead is removed from the wastewater before discharge the benefit will be only partial. The net effect on reducing mean daily lead intake and mean blood lead will be slight, a few tenths of a μg/dl from the normal 2-3 μg/dl attributed to drinking water.

Thus, the sum total of the reductions in blood lead which will accrue to the general public from proposed or recently enacted limitations can be estimated to be in the range of 0.5-1.0 μg/dl, a reduction of approximately 2 1/2 to 5 per cent from a present mean of about 20 μg/dl. The banning of paint containing more than 0.06 percent lead by The Consumer Product Safety Commission (1977) will virtually eliminate the danger of childhood lead poisoning from the ingestion of paint hereafter produced and applied around the home. However, this limitation does nothing for the hazard from ingestion of high-lead paints such as were applied in previous years, and which are still the principal cause of lead poisoning in children, and will have essentially no effect or reducing present average blood lead values of children now so exposed. (Agencies such as Housing and Urban Development might be able to devise and implement limitations which would attach this problem, which is basically a housing problem).

Thus, while all of the above are helpful and desirable steps to limit exposure to lead, they will increase the margin of safety of the general population only slightly; more is desirable.

Consideration of Section 8 shows that at low air lead concentrations (as will soon exist generally, with the exception of occupational exposure and possibly the immediate vicinity of major point sources) the principal source of lead for the general population is through ingestion of food. For most individuals, food contributes 60 percent or more of total absorbed dose, and if the mean blood lead concentration of the general public is to be lowered, reduction of dietary lead appears to be the most logical means of approaching the problem.

In summary, there is a need for additional reduction in human lead intake, and food is the one large contributor offering the greatest promise for achieving additional significant reductions affecting the general population.

In Section 8, the present levels of lead in foods, fresh and processed, were analyzed; the potentials for reductions were developed, and the benefits such reductions can bring in reducing mean blood leads and the number of persons with blood lead above any given level were described. Possible approaches to limitations, both food and non-food, are briefly described and the benefits and disadvantages of each are analyzed in Section 9.3, and a case study of food canning processes is examined in Section 9.5.

9.3 APPROACHES TO LIMITATIONS

9.3.1 Criteria For Prioritization

Theoretically, any use at all of lead should be the starting point for consideration of possible limitations on its use. However, lead, a metal that has been used by man for thousands of years, has too many valuable and essential uses to make its total abandonment a viable option, i.e. total prohibition is not a feasible limitation. There are technically feasible options or alternatives for many, if not most of the dissipative uses of lead, and it is theoretically possible to prohibit those uses, if this is the most desirable solution to achieve exposure reductions.

There is no definitive evidence of demonstrable adverse biological effects on the general adult population of the United States due to exposure to lead, except in limited areas in the proximity of major point sources, such as smelters, (except, of course, unusual situations such as illicit whiskey or poorly-fired hand-crafted drinking utensils). Part of the reason for this may be that there is not definitive evidence that lead has significant teratogenic, mutagenic, or carcinogenic properties in man. Another reason is that the evidence to date indicates that lead does not bioconcentrate or biomagnify in man's food chain; and plants are not adversely affected at present mean concentrations of lead in the atmosphere and in the soil.

On the other hand, there is no evidence that lead is an essential element, even though it has been part of the body burden of man for centuries. Also, the proof is conclusive that lead is a toxic element. Lead compounds do not biodegrade to non-toxic residues; the lead moiety can persist infinitely, and there is no form of lead which is non-toxic.

Proceeding from these basic premises, it is possible to establish a set of criteria for prioritizing potential limitations on lead. The criteria utilized in this investigation included the following, not necessarily listed in order of importance, for considering the various ways in which man is exposed to lead and how these might be limited:

- Fraction of man's total absorbed dose attributable to that cause
- Directness of exposure to man or biota from source in question
- Dissipative or non-dissipative use
- Quantity of lead released to the environment
- Bioavailability of released lead
- Number of individuals who would benefit from the limitation
- Special benefits of the limitation to particularly sensitive segments of the general population
- Availability of alternatives for the use or process limited
- Technical and economic feasibility of appropriate limitations
- Cost of instituting limitation vs. health benefits derived
- Cost of monitoring and enforcement of limitation
- Absence of Existing limitations

9.3.2 Prioritization Of Possible Limitations

On the basis of the above criteria and the information described on human health effects (Section 7) and the nonoccupational exposure of humans to lead (Section 8) these sources of lead exposure were identified as those most meriting consideration of possible limitations, in the following approximate order of priority:

- Leaded gasolines (including contaminated waste lubricating oil)
- Adventitious lead in foods
- Lead in drinking water
- High-lead paints in homes
- Lead-based paints
- Lead in printing inks
- Emissions from lead industry processes

- Lead ammunition
- Lead in plastics and rubber
- Emissions from indirect sources
- Lead-acid storage batteries
- Miscellaneous uses of metallic lead

These sources are discussed in the suceeding paragraphs, in terms of the criteria listed above. An evaluation of the importance of each type of source in terms of the individual criteria is helpful in prioritizing the consideration and development of possible limitations, even though such an evaluation is necessarily somewhat subjective. The importance of each factor was assessed as major (large), moderate, or minor (small), signified by a +, 0, or - sign. The rating scale had to be reversed for negative factors, such as the cost of monitoring and enforcement. More weight was given to the factors determining the degree of risk posed by the particular use. Although the ratings are not numerically additive a general ordering of the need and feasibility of limitations is possible, and a high score is suggestive of a need for a high priority ranking. The evaluation is summarized in tabular form in Table 9.2.

Not surprisingly, leaded gasolines and adventitious lead in foods had a preponderance of +'s and the highest scores of all. Also scoring high were high-lead paints in place, printing inks, and emissions of lead from lead industry processes.

A key final judgement factor is one presence or absence of limitations. As discussed later in this chapter many limitations to control exposure to lead already exist.

9.3.2.1 Leaded Gasolines--

As pointed out in Section 5.2.2.2, upwards of 120,000 metric tons of lead were dispersed to the environment in 1975 from the consumption of leaded gasolines. Most of this was to the atmosphere, with a large portion of the balance reporting to the waste lubricating oil. Some of this was burned, some was discarded to sewers or to solid waste, and some was re-refined.

The quantity released was so enormous (far more than all other annual releases combined) that it was assigned a + + ranking. It is a totally dissipative use of lead, emitted in a form with high bioavailibility throughout the United States, and exposing the entire population. Alternatives are available, and limitations are technically and economically feasible. The cost of monitoring and enforcement is categorized as moderate. The entire population will benefit from limitations on the use of leaded gasolines, including the susceptible population segment of children, especially inner-city children. Accordingly, all of these factors were rated as +'s.

TABLE 9.2 ASSESSMENT OF FACTORS CHARACTERIZING A NEED AND FEASIBILITY OF LIMITATIONS ON LEAD

Assessment Factor	leaded gasoline	adventitious lead in food	lead in drinking water	high-lead paint in place	lead-based paints	printing inks	lead-acid storage batteries	lead in plastics and rubber	ammunition	misc. uses of metallic lead	emissions - lead industry processes	emissions - indirect sources
Fraction of dose	0	+	-	+		-	-	-		-	0	0
Directness of exposure	+	+	+	+	-	+	-	-	+	-	0	0
Dissipative vs. nondissipative	+	+	+	+	+	+	-	+	+	0	+	+
Quantity released	++	-	-	-	-	-	0	0	0	-	+	0
Bioavailibility	+	+	+	+	+	+	-	-	+	-	+	+
No. of individuals benefited	+	+	-	0	0	-	-	-		-	-	-
Spec. benefit susceptible pop.	+	+	0	+	+	0	-	-		-	0	-
Availibility of alternatives	+	+	+	-	+	+	-	0	0	-	0	0
Technical and economic feasibility	+	+	+	-	+	+	-	0	+	-	0	0
Benefit/cost ratio	0	+	+	-	0	0	-	0	0	-	-	-
Low cost monitoring and enforcement	0	+	0	-	0	0		+	0	+	-	-
Absence of existing limitations	0	+	0	+	0	+	+	+	0	+	-	0

[a] Source: Battelle - Columbus estimate

[b] Symbols
+ large, major
0 moderate
- small, minor

The combination of the above factors has, of course, already prompted the placing of strict limitations on leaded gasolines, as discussed elsewhere in this report (Section 5.2.2.2, 8.2.3 and 9.4.1). As noted therein, the phasedown to an average gasoline content of 0.5 g/gal by 1980 is predicted to reduce the mean national air lead concentration to about 0.3 μg/m^3. At this concentration the contribution of leaded gasoline to the average absorbed dose will be about 0.2 μg/dl, approximately 1 percent of the mean blood lead of U.S. adults. Present dose is considered to more properly fall in the moderate category (rated 0), especially in high-traffic urban areas.

This very large reduction in the use of leaded gasolines will concomitantly automatically reduce the dispersion of lead to the environment through the disposal of waste lubricating oil.

There will, however, still be a large release of lead to the environment from the combustion of gasoline, possibly 40,000 to 45,000 metric tons per year in 1980-1985. Thus, it is conceivable that additional limitations on leaded gasolines might be indicated. One approach might be to extend the no-lead fuel requirement to trucks and piston-engine aircraft, now exempt from the restrictions which have been placed on new passenger cars.

Limitations could be placed on the disposal of waste lubricating oil, but the operation, monitoring, and enforcement of such limitations could pose almost insuperable problems, as well as being extraordinarily costly.

As evident from the projected ambient air concentrations which will be attained upon completion of the phasedown, the possible additional reduction in absorbed dose for individuals from further limitations is estimated to be slight.

The subject of additional limitations on leaded gasolines is extremely complex, and the assemblage and analysis of the voluminous data which would be required to validate any recommended changes was beyond the scope of this investigation. On the basis of the basic parameters outlined above, the conclusion is offered that there is no pressing need for additional limitations in this area at this time with a recommendation that the results of the phasedown be awaited and evaluated before further action is taken.

9.3.2.2 Adventitious Lead in Food and Beverages--

As discussed in Sections 8.5 and 8.6, most of the intake of lead for the general adult population is from food, and as the lead phasedown proceeds and ambient air lead concentrations diminish, this will become the predominant source. A major source in food is canned food. At present from one-half to two-thirds of the lead content of canned food is adventitious, arising from the lead in the solder used to form aseptic seals in the side seams of the cans. In testimony at the EPA hearing on the proposed national ambient air quality standard for lead, the acting head of the FDA Bureau of Foods estimated that about two-thirds of the lead in canned food is from the solder, with the remainder in the food itself prior to canning. It was further estimated that for a two-year old child, with a total average daily dietary lead intake of 98 μg, about 18 μg or 18 percent comes from food packed in cans (Roberts, 1978). Thus, a significant fraction of the dose arises from this cause, and the exposure route is direct, causing both of these factors to be rated as +'s.

Much the same relationship exists for adults; since canned foods, on average, constitute about 30 percent of U.S. diets, upwards of 15 percent of daily lead intake from food could be eliminated.

Preliminary analysis showed that adventitious lead in foods had a priority ranking comparable if not greater than that of lead in gasoline, and probably merited the development and adoption of limitations more than any other candidate. Alternatives are available (+), with satisfactory technological and economic feasibility (+), and the benefits are large for the costs involved (+), costs of monitoring and enforcement would be moderate to low (0), the entire population will benefit from reduced lead in foods (+), particularly the specially sensitive group, children (+). This limitation candidate had 10 + factors, more than any other category, indicative of its importance in considering prioritization of lead limitations.

Further discussion on the form and nature of such possible limitations and their technological feasibility is deferred to Section 9.5, devoted to this topic. It is hoped that this analysis will provide further confirmation and support to the Food and Drug Administration's efforts already underway on this problem.

9.3.2.3 High-Lead Paint in Homes--

The special problem of high-lead paint in homes has been alluded to earlier. This is acknowledged to be the principal cause of childhood lead poisoning, probably affecting several hundred thousand children, peimarily those in substandard and deteriorated housing in the cities of the U.S. Since at the time these lead paints were applied, in the years prior to WW II, lead contents of the paint were characteristically as high as 60 percent or greater, even a few chips of paint eaten by a child will produce unacceptable doses. For these children the fraction of dose from this cause can be overwhelming, and obviously receives a + rating. The problems arise from ingestion, which is a totally direct route (+).

As noted in Section 4.3.3, it has been estimated that several million tons of lead-based paints have been applied to houses, a dissipative use (+). As Chisolm (1977) has noted, there were, according to the 1970 U.S. Census, 30 million dwelling units built before 1950 and still in use. It is generally estimated that at least 90 percent of this old housing stock contains lead-pigment paints in areas accessible to children. As discussed in Section 7.4.3, Gilsinn (1972) has estimated on the basis of census data that approximately 7,000,000 housing units are old enough to contain paint made with high concentrations of lead, and that as many as 600,000 children in 241 standard metropolitan statistical areas (SMSA's) had elevated blood lead levels (>40 µg/dl). While this is numerically but a small fraction of the total population, the risk is high.

Although the tonnage of lead paint which has been applied to homes is in the millions of tons, the rate of release of flaking lead paint and lead-containing plaster released to the environment is relatively quite small (-).

Nevertheless, the location of the releases and their concentrated nature give them a significance far beyond their quantities. Since the entry to the body is by ingestion, the route of exposure is direct, (+), and the risk is compounded by the high absorption of children. The bioavailibility is reportedly less for lead in a paint film than for lead in some of the soluble lead salts, but it is quite high enough to have caused numerous cases of lead poisoning in children, and is rated +.

However, this situation is another facet of the "resource in use" category comprising the huge inventory of millions of tons of lead already in place, and is one which will not be effectively controlled by limitations on future lead use.

There are few satisfactory alternatives (-). Elimination of the problem by immediate demolition of the affected housing and its replacement by new lead-free housing does not appear to be an economically feasible solution (-). A process of applying a heat-set shrink film has been developed to cover and effectively seal off painted interior woodwork (Mirick and Nowacki, 1977), but the economics and practical feasibility of this approach is doubtful.

A simple and sure solution to the problem of high lead paint and plaster (and the concomitant high lead contents of the associated house dust) in old and deteriorated substandard housings has not been devised. Some legislation does exist, e.g. HUD's regulations covering lead paint in Federally-owned or assisted housing (See Section 9.4.1.15), but this legislation does nothing for non-Federal housing.

Some states, e.g. Massachusetts, have enacted legislation which requires removal or cover of paint, plaster, or other accessible materials containing dangerous levels of lead in residential premises where a child under 6 years of age resides. Painting over with non-lead-based paint does not constitute compliance. As a result of this Act, it is estimated that during the 3-year period 1975-1977, approximately 10,000 units were deleaded in Massachusetts (which represents, however, only 3.3 percent of the 300,000 units throughout Massachusetts still to be deleaded) (Feldman, 1978). Even this solution is not without problems. In the effort to protect children from the hazards of lead paint, some of those removing it are falling victim. Unsafe deleading procedures are resulting in cases of lead intoxication among the deleaders themselves (Feldman, 1978).

It is not clear how present-day limitations can overcome the adverse effects of lead paint applied to houses perhaps 40 or more years ago. Some possible non-structural approaches to mitigating the child poisoning problem are discussed in Section 9.3.4.

9.3.2.3 Lead-Based Paints--

The Lead-Based Poisoning Prevention Act (LBPPA) defines as lead-based any paint containing more than 0.06 percent lead and their use is banned as a hazardous product in situations where there is a reasonable probability that children would have access to them, i.e. around the home. Metal furniture was subsequently exempted from the ban, and The Consumer Product Safety Commission also determined that paints used industrially were not "consumer products" and hence were not within the scope of the LBPPA.

The lead exposure hazard of lead-based paints is only to the childhood segment of the general population; the exposure to adults is minimal. (There can be major risks to painters, scrap yard employees, and ship breakers, but these are occupational problems, excluded from this analysis. OSHA has addressed these problems in its latest regulations).

The recycling of painted steel scrap to steel furnaces is a contributor to atmospheric lead emissions, but limitation of such emissions is better controlled as a part of steel industry regulations than as a seperate category. The reduction in average individual absorbed dose from such limitation would be expected to be small; the benefits would accrue principally to residents in the proximity of such furnaces.

Lead-based paints were rated as +'s as a dissipative use, with a high bioavailibility, with technically and economically feasible alternatives available, and with special benefits occruing to the susceptible childhood segment of the population by their limitation. However, since lead has effectively been banned for uses where children might be harmed, the problem is a most one with respect to any need for additional limitations.

9.3.2.5 Printing Inks--

The brilliant colors provided by lead chromate pigments are utilized in colored printing inks to a certain extent. Chief use appears to be for colored pictures in magazines and books and in the gaily-colored gift wrapping papers and foils. (The Consumer Products Safety Commission is now investigating this area).

Tonnage data were not identified, but only a fraction of the 15,000 metric tons of pigment colors consumed in 1976 went to printing inks. Ultimate fate of most printed matter appears to be a landfill or an incinerator; the latter would contribute slightly to air emissions of lead.

The chief concern with printing inks is the exposure to small children who might chew paper, one form of pica. This is presumed to be a less common occurrence than chewing woodwork, and the lead content of printed colored paper is only a fraction of that of lead-based paint; 0.12 percent was found by Campbell (1976). This is only twice the 0.06 percent allowable content of "lead-free" paints, and appears to present a minimal risk.

Printing inks received + ratings for directness of exposure (through ingestion), a high bioavailibility, and as comprising a dissipative use. However, the fraction of dose estimated to result from this cause was rated as minor, and alternatives are available which are technically and economically feasible.

A limitation on the use of lead pigments in inks used for children's books might be considered, but the reduction in individual absorbed dose would probably be imperceptible. This appears to be a low-priority candidate for limitation.

9.3.2.6 Lead-Acid Storage Batteries--

Lead-acid storage batteries represents the largest single use of lead (upwards of 750,000 metric tons of lead were consumed in 1976), and annually consumes more than half of total consumption. However, it also represents an outstanding example of an essentially non-dissipative use, not exceeded by any other base metal. The lead in a storage battery is used in a closed container, with no losses to the environment during use. The exposure of the general public to lead from the use of batteries is virtually nil, and no dose benefits would accrue if lead batteries were replaced by non-lead battery substitutes. There is no apparent alternative or technically and economically feasible substitute for the lead-acid battery. Noteworthy developments in battery technology include the current trend to the maintenance-free lead-calcium battery in place of the older lead-antimony type.

On a longer-term basis other battery systems are in the research and development stage. Of these candidates, the zinc-air battery is judged to be the one most likely to approach the lead-acid battery in its present applicability to existing needs, but practical commercial forms of this battery are projected to be at least five years away, with a price several times higher than lead-acid storage batteries. Other lead-free systems are being investigated, but it is estimated that it may be 10 to 15 years before any of these could successfully replace lead storage batteries.

The above discussion considered the lead exposure hazard resulting from the use and application of lead-acid storage batteries and found it to be insignificant. The industrial processes associated with this endeavor; battery breaking, secondary smelting of old batteries, oxide production, and pasting of battery plates, are another matter. Significant air and water emissions of lead are possible, and regulation of the operations is needed to prevent creation of lead exposure hazards. However, these are the same problems encountered throughout the lead processing industry, and are not unique to storage batteries. These are better considered all together as process emissions problems (see Section 9.3.2.10).

Lead-acid storage batteries ranked very low as a candidate for limitations (See Table 9.2). This is a non-dissipative use, with a biologically non-available form of lead, releases almost none to the environment, has no technically or economically feasible alternatives, and even rigorous limitations on the use of lead storage batteries would do essentially nothing towards reducing average lead intakes.

9.3.2.7 Lead in Plastics and Rubber--

Lead stabilizers find use in plastics, primarily PVC, e.g., for electric wire insulation, to protect the plastic from the heat necessary for their processing and forming (this use consumed about 8,000 metric tons in 1975 (see Section 5.2.2.4). This is a wholly dissipative use (+); none of it is recycled. The bioavailibility of the lead in the plastic is low (-), the likelihood of a direct exposure through ingestion of such items is also low (-). The fraction of the average individual's dose attributable to this source is estimated to be unmeasurable (-).

The lead-containing plastic items (perhaps 1-2 percent lead) are eventually discarded to waste, probably either to a landfill or to an incinerator, analogous to the fate of paper. In the one case lead could leach out to groundwater, and comprise an atmospheric emission in the other. No information was identified on the resultant health hazards; in the absence of definite information the judgement is made that the quantities so released would be small to moderate (0) and that they would not constitute major lead exposure hazards.

Some alternative stabilizers are available for this use, primarily tin compounds, which may also pose their own risk (0). Overall this application did not appear to warrant a high priority for consideration of limitations.

9.3.2.8 Lead Ammunition--

Two problems have been identified with respect to the environmental effects of lead ammunition, one widespread and one quite unusual and rare. The use of lead shot has been proven to produce serious lead poisoning in waterfowl, causing the death of up to 2.4 million annually, excluding sublethal effects that make millions of additional birds more vulnerable to disease and predation (Carter, 1977). This situation obviously called for some kind of limitations, not because of human health implications, but for the public welfare.

In response to this need, the U.S. Fish and Wildlife Service has instituted a prohibition against the use of lead shot in the flyways most affected; steel shot has been substituted for lead shot.

The unique case concerned employees at an indoor pistol range who developed clinical symptoms of lead poisoning, due apparently to excessive air lead concentrations. No reports of similar cases have been identified, and this case is so unusual that it would appear to have only a low priority with respect to candidacy for environmental limitations. Perhaps the only limitation needed would be an extension of OSHA regulations on occupational exposure to lead to such establishments.

9.3.2.9 Miscellaneous Uses of Metallic Lead--

Metallic lead is employed for a variety of uses utilizing its high density and corrosion resistance, and for many of these uses there are few practical substitutes. Radiation attenuation, ballast, sound absorbers, tire balancing weights, lead pipe, and flashings are a few examples of such uses (see Section 4.3.5 and 5.2.2.5).

Although there are few data on the recycle of lead from such uses, as a generalization it may be observed that the extent of retrieval and recycle is inversely proportional to the diffuseness of the use and directly proportional to the mass of the individual pieces. Thus, recycle may be relatively high for large items used industrially, and near zero on little items widely scattered, such as tire balancing weights. Recycling is basically a function of economics, and will improve as it becomes more economically attractive, either by natural increases in lead prices, or by the incorporation of some subsidy arrangements.

However, the alternate destination besides recovery and recycle is most probably to a landfill, from which its dispersal to the environment would be negligible because of its chemically inert metallic form.

The factors in this category were characterized by - and 0 ratings (Table 9.2) and the need for investigation of possible limitations was assigned a low priority.

9.3.2.10 Emissions from Lead Industry Processes--

One non-negligible source of exposure to lead and its compounds is the lead industry and its processes (along with lead emissions encountered in processing other nonferrous base metal ores). These exposures arise primarily from lead-containing air and water emissions. These will be found at lead mines and mills, primary and secondary lead smelters, storage battery plants, and pigment plants.

The present status of limitations on lead producing and consuming industries is described in some detail in Section 9.4, and it is evident from the variety of these that a high priority has been assigned to controlling these emissions. Since this has been a continuing effort for a number of years most of the major lead exposure problem areas have been taken care of. Effluent guideline limitations remain to be promulgated for lead battery manufacturing, and air emission standards specifically regulating lead emissions from existing smelters, etc., have not yet been developed. The newly-promulgated ambient air lead air quality standard for lead of 1.5 $\mu g/m^3$ (U.S. Environmental Protection Agency, 1978), will no doubt have the indirect effect of controlling air emissions from these existing sources.

The acquisition of the detailed experimental and field data on emissions, required to establish a basis for proposing additional limitations, was beyond the scope of this program. No further recommendations can be offered until the impacts of recent regulations are observed. Overall, the area of lead emissions from lead producing and consuming industries appears to be fairly well in hand.

9.3.2.11 Emissions from Indirect Sources--

A number of so-called indirect sources, not part of the lead industry, are emitters of lead, primarily to the atmosphere. These are sources where lead is present as a minor constituent, as in coal, fuel oil, or cement rock, or as one component of a waste, as in solid waste or sewage sludge (see Section 5.2.3). As noted therein, and tabulated in Table 5.1, these do not comprise major point sources of lead emissions, although the sum of all such emissions is estimated at several thousand tons. Lead-painted steel scrap accounts for an indeterminate fraction of the lead emitted from iron and steel furnaces. Discarded lead-bearing materials, e.g. colored magazine paper, lead containing rubber or plastics, waste lubricating oil, solder from tin cans, tire weights, lead toys, collapsible tubes, lead storage batteries, etc., account for the approximately 1,200 metric tons emissions estimated for the incineration of solid wastes (Section 5.2.3.4).

These sources are fairly diffuse geographically, and vary widely in performance and emission characteristics. Limitations have been promulgated for a number of the types of sources based on control of particulates, which are regulated on a "process-weight" basis. Since lead is emitted as a particulate, these regulations afford a fair degree of control of lead emissions. The detailed experimental and field emission data needed to propose additional limitations on lead emissions was not available to this investigation, and no recommendations for additional limitations can be offered. The 1.5 $\mu g/m^3$ ambient air lead standard may provide all the control needed for these sources. In total, they contribute little to the average individual lead intake, and additional limitations from point sources of this type are not regarded as high priority items.

However, one other type of indirect source which has resulted in the dispersion of lead and lead compounds to the environment is solid waste disposal. One facet of this is the disposal of wastes of all sorts to landfills; this has been largely uncontrolled in the past, and the Resource Conservation and Recovery Act of 1976 (RCRA) was enacted in response to this need. Another facet is the disposal of sewage sludge to agricultural lands. Such sludges may contain lead and other heavy metals, cadmium being the one of most current concern, and criteria are needed for these heavy metals.

9.3.4. Non-Structural Approaches

The inapplicability of new limitations on lead use, handling, and disposal to mitigate the very serious problem of lead poisoning of children living in substandard and deteriorated housing painted with high-lead paints was alluded to in Section 9.3.2.3. This historical problem may well continue until the lead-containing substandard housing is eventually replaced. However, immediate demolition and replacement is probably an unattainable solution because of the costs. It may also be impractical and near-impossible to identify and eliminate all sources of exposure for these children from product use or environmental sources, even with stringent controls on lead.

An alternative less-costly solution is available which should very nearly attain the same objective. The screening of infants and young children, with appropriate follow-up examinations, can identify the existence of an incipient problem, before irreversible damage occurs, so that corrective measures can be promptly instituted. The effectiveness of this approach in eliminating childhood deaths from lead poisoning and in minimizing childhood lead poisoning cases has been demonstrated in several cities, for example, Baltimore (Lutz, et al., 1970) and in Chicago (Sachs, et al., 1970).

On the basis of these and other results demonstrating the effectiveness of this approach for the control of childhood lead poisoning it seems likely that intensified surveillance of the population at risk, through local lead poisoning prevention and treatment centers throughout the nation, probably offers the most practical method to very significantly reduce, if not virtually eliminate, the adverse effects of exposure to lead of this most sensitive segment of the population to the extreme risk of acute lead intoxication. It should be emphasized that the adverse effects in question are not threshold effects of lead below traditional levels of concern, as discussed in Section 9.2.1, but are those associated with clinical lead poisoning.

Accordingly, the health benefits to be so gained should significantly exceed those accruing to this particularly sensitive population group from any other limitation on lead including the 1.5 $\mu g/m^3$ ambient air lead standard, or from any of the limitations on adventitious lead entering food.

Thus, while this surveillance and treatment is not a limitation on lead, per se, its potential health benefits are so large that it is strongly recommended that the potential of additional local, state and Federal funding of this approach be very seriously considered. Because such a substantial stock of old lead-painted housing is still in use in this country, the need for screening and pediatric management in high-risk areas will continue for some years. The benefit: cost ratio for this approach is believed to be as high or higher than any other action which has been instituted or proposed for consideration.

Non-structural approaches can be applied to other areas, for example, with proper labelling, lead-containing paints were exempted for certain uses under the Lead-Based Paint Poisoning Prevention Act (See Section 9.4.1.14).

9.4 EXISTING AND PROPOSED LIMITATIONS

More than thirty existing or imminent Federal regulations governing the manufacture, use, or disposal of lead and lead-containing compounds have been identified. These fall into two broad general categories: direct regulations specifically governing lead, and indirect regulations governing some associated pollutant, the control of which has a high probability of also controlling lead emissions. (Particulate regulations are a good example of the latter type).

Another way of categorizing existing limitations is whether they govern a process, a product, or the quality of the environment. Most of the regulations which derive from the Clean Air Amendments of 1970 (PL 91-604) and the Federal Water Pollution Control Act of 1972 (PL 92-500) control processes which produce air or water lead emissions through New Source Performance Standards (NSPS) and Effluent Guidelines. A smaller number of regulations control products which do or can contain lead, and the final group sets limits on lead concentrations in air or water.

The known existing limitations are summarized in Table 9.3, and described and discussed in the following pages. The table indicates the mode of control, the numerical value of the limitation, the Federal agency exercising control, and the section of the Code of Federal Regulations (CFR) containing the official regulation (or Federal Register, if the regulation or proposed regulation is not yet in the CFR).

At the conclusion of the section some of the recently-enacted enabling legislation is briefly discussed, which does not specifically apply to lead and its compounds either directly or indirectly, but which has the authority

TABLE 9.3 SUMMARY OF EXISTING AND PROPOSED FEDERAL REGULATIONS GOVERNING LIMITATIONS ON LEAD.

Activity Controlled	Mode of Control	Limitation[a,b]	Agency Exercising Control	Citation
		Direct Limitations		
Nonferrous ore mining and milling	Effluent guidelines	0.3 mg/l Pb	EPA	(40 CFR 440.20) (43 FR 29771)
Ferroalloy ore mining and milling	Do	0.2 mg/l Pb (mining) No limitation (milling)	EPA	40 CFR 440.40
Primary lead	Do	No discharge	EPA	40 CFR 421.70
Primary copper	Do	No discharge	EPA	40 CFR 421.40
Electroplating	Do	80 mg/sq m plated	EPA EPA	40 CFR 413.12[c] 43 FR 6560[d]
Lead battery manufacture	Do	Regulations not yet promulgated	EPA	- - -
Chlorine-caustic	Do	0.0025 kg/kkg product	EPA	40 CFR 415.60
Lead monoxide	Do	No discharge	EPA	40 CFR 415.440
Chrome pigments	Do	0.14 kg/kkg of product	EPA	40 CFR 415.340
Television picture tubes	Do	4.5 g/kkg glass (BPT) 0.45 g/kkg glass (BAT)	EPA	40 CFR 426.110
Hand pressed and blown glass	Do	No limits (BPT) 0.1 mg/l (BAT)	EPA	40 CFR 426.130
Lead-sheathed rubber hose	Do	0.007 kg/kkg raw material (BPT & BAT)	EPA	40 CFR 428.20
Gasoline antiknock additives	Air programs	0.8 g/gal 1/1/78 0.5 g/gal 10/1/79	EPA	40 CFR 80.20
Lead-based paint	Lead content	0.06 percent Pb	CPSC	(16 CFR 1303.) (16 CFR 1500.17)
Lead-based paint on housing	Lead content + other	0.06 percent Pb Elim. in existing housing	HUD	24 CFR 35.
Occupational exposure	Exposure standard	50 $\mu g/m^3$	OSHA	29 CFR 1910.1025
Community water systems	Lead content	50 $\mu g/l$	EPA	40 CFR 141.11
Evaporated milk	Lead content	0.3 ppm tolerance (proposed)	FDA	39 FR 42740
Ambient air standards	Air programs	1.5 $\mu g/m^3$	EPA	40 CFR 50.12
Lead-decorated glassware	Voluntary Standard	50 ppm leachable from lip and rim	EPA, FDA, CPSC	Under consideration

(Continued)

TABLE 9.3 (Continued)

Activity Controlled	Mode of Control	Limitation[a,b]	Agency Exercising Control	Citation
		Direct Limitations		
Lead arsenate residues on food	Lead content	7 ppm Pb	EPA	40 CFR 180.194
Duck hunting	Lead shot	Some prohibition	DOI	50 CFR 50 CFR 20.134
Ocean dumping	Dumping permits	Permit required	EPA	40 CFR 227.
		Indirect Limitations		
Primary lead smelters	NSPS	50 mg/dscm (particulates) 0.065 percent SO_2	EPA	40 CFR 60.180-.186
Primary copper smelters	NSPS	50 mg/dscm (particulates) 0.065 percent SO_2	EPA	40 CFR 60.160-.166
Secondary lead smelters	NSPS	50 mg/dscm (particulates)	EPA	40 CFR 60.120-.123
Ferroalloy production facilities	NSPS	0.23-0.45 kg/MW-hr (particulates)	EPA	40 CFR 60.260-.266
Electric arc steel furnaces	NSPS	12 mg/dscm (particulates)	EPA	40 CFR 60.270-.275
Incinerators	NSPS	0.18 g/dscm (particulates)	EPA	40 CFR 60.50-.54
Sludge incinerators	NSPS	0.65 g/kg dry sludge (particulates)	EPA	40 CFR 60.150-.154
Portland cement manufacture	NSPS	0.15 kg/kkg of feed (particulates)	EPA	40 CFR 60.60-.64
Gasoline vapor recovery	Air programs	90 percent recovery (stage I) 0.4-0.6 g/gal (Stage II)	EPA	
Petroleum liquid storage	Air programs	Floating roof or equivalent	EPA	40 CFR 60.112

[a] Effluent guideline limitations given are 30-day averages.

[b] Abbreviations:

BPT - Best practical control technology currently available
BAT - Best available control technology economically achievable
NSPS - New Source Performance Standards
dscm - Dry standard cubic meter

[c] Electroplating effluent guidelines standards suspended December 3, 1976.

[d] Pretreatment standards reproposed February 14, 1978.

to set such regulations and may do so at some future date when fully implemented.

9.4.1 Direct Limitations on Lead

Direct limitations include those governing water and air emissions from the various manufacturing segments of the metals industries, and use-related emissions; those establishing limits on lead concentrations in air or water, and those governing disposal.

Emissions to waterways are governed by Effluent Limitations Guidelines, established under the authority of the Federal Water Pollution Control Act amendments of 1972 (P.L. 92-500). By the terms of the Act, two levels of control are to be achieved; initially the "best practical control technology currently available" (BPTCA, or colloquially, BPT) and at a later date a higher level of control designated "best available technology economically achievable" (BATEA, or, colloquially, BAT). BPT was to have been achieved by July 1, 1977 and BAT by July 1, 1983, but for a number of reasons these dates have been extended.

Effluent guidelines may be established for existing sources, for new sources, and for pretreatment before discharge to a publicly-owned wastewater treatment plant. Another characteristic of Effluent Guidelines Limitations is that they are source-oriented and control emissions from point sources; the quality of the receiving body of water is not a factor in the limitations.

The scope of air emission regulations is narrower than those for aqueous effluents. These are promulgated under the authority of the Clean Air Act, as amended in 1974, and as further amended by the Clean Air Act Amendments of 1977 (P.L. 95-95). Federal standards apply only to new sources: standards for existing sources are set by the states.

9.4.1.1 Nonferrous Ore Mining and Milling--

Under Ore Mining and Dressing, Subpart B, Base and Precious Metals Subcategory (40 CFR 440.20) effluent limits on lead have been set on both mine drainage and effluents from froth flotation mills at (0.3 mg/l)*. These limitations cover mines operated to obtain copper-bearing ores, lead-bearing ores, zinc-bearing ores, gold-bearing ores, silver-bearing ores, or any combination of these ores from open-pit or underground operations. The limitations are for BPT; no BAT limitations have yet been promulgated. These final limitations just recently issued (43 FR 29771) supersede the interim final regulations and slightly adjust lead concentrations.

As a general rule, large-scale, outdoor-type, indurtrial and mining operations, such as the ferrous and nonferrous industries, are also granted a variance to discharge untreated overflow in excess of the quantity resulting from specified maximum storm events, generally in the case of BPT defined as the 10-year, 24-hour precipitation event.

---

*Values cited in this section for effluent guidelines are the 30-day averages not to be exceeded. Daily maximums are higher, most frequently by a factor of 2.

9.4.1.2 Ferroalloy Ore Mining and Milling--

Subpart D, the Ferroalloy Ores Subcategory of the Ore Mining and Dressing Category, (40 CFR 440.40) places lead effluent limits on mines producing more than 5,000 metric tons or more of ferroalloy ores per year. Ferroalloy metals include chromium, cobalt, columbium, tantalum, manganese, molybdenum, nickel, tungsten, and vanadium. Effluent limits (BPT) for mine drainage for lead is 0.2 mg/l. There are no limits on effluents from flotation mills. The ferroalloys are characteristically high-value, low-tonnage metals, and mine tonnages are minor, compared to those of copper, lead, and zinc. For example 1975 tonnages of lead and zinc ores were 7.8 and 5.7 million metric tons, respectively (Section 5.2.1.1). Losses of lead to effluents for the ferroalloy ores are correspondingly minor.

9.4.1.3 Primary Lead--

Subpart G, the Primary Lead Subcategory of the Nonferrous Metals Manufacturing Point Source Category (40 CFR 421.70), includes the production of lead at primary lead smelters and refineries, but primary lead refineries not located on-site with a smelter are not part of this subcategory.

BPT limitations are for "no discharge of process waste water into navigable waters". There are two exceptions. If an impoundment is designed to contain the 10-year, 24-hour precipitation event, process waste water equal to the excess collected in such an event may be discharged. Discharges may be made monthly of the excess of precipitation in the impoundment above evaporation, except that the lead content of such discharges shall not exceed 0.5 mg/l.

BAT limitations are analogous to the BPT limitations, the principal difference being that the storm event is increased to the 25-year, 24-hour event.

9.4.1.4 Primary Copper--

Subpart D of the Nonferrous Metals Manufacturing Point Source Category covers the Primary Copper Subcategory (40 CFR 421.40). As with lead, primary refineries not on site with primary smelters are not included; in the case of copper refining, there is a separate subcategory to cover these facilities.

Limitations on lead discharges from primary copper operations are the same as for primary lead; no discharge except for excessive precipitation, and routine discharges of such excess are limited to 0.5 mg/l lead, except for maximum storm events.

9.4.1.5 Electroplating--

Limitations on lead discharges are specified only for the Electroplating of Common Metals Subcategory (Subpart A) of the Electroplating Point Source Category (40 CFR 413.12). Omitting some of the minor details, lead discharges are not to exceed 80 mg per square meter per operation. Since areas of irregular surfaces are essentially indeterminate, other parameters such as ampere-hours used in plating can be used, after calibration.

Effluent guidelines regulations were originally promulgated April 24, 1975 (40 FR 18130-18148). These were challenged by the industry, and the common metals subcategory regulations were suspended December 3, 1976 (41 FR 53018). Proposed pretreatment standards for existing sources were re-proposed February 14, 1978, (43 FR 6560-6573), and are still in the comment period.

9.4.1.6 Lead Storage Battery Manufacture--

Work is in progress on the establishment of effluent guidelines for the manufacture of lead storage batteries, but no information on limits is yet available.

9.4.1.7 Chlorine-Caustic--

Subpart F of the Inorganic Chemicals Manufacturing Point Source Category covers the Chlorine and Sodium or Potassium Hydroxide Production Subcategory (40 CFR 415.60). Plants using the diaphragm cell process for the manufacture of chlorine and caustic are limited to lead discharges of not greater than 0.0025 kg/kkg of product (2.5 g/metric ton) to meet BPT guidelines. Guidelines for BAT are "no discharge".

9.4.1.8 Lead Monoxide--

Lead monoxide (litharge) manufacture is covered by Subpart AR, Lead Monoxide Production Subcategory of the Inorganic Chemicals Manufacturing Point Source Category (40 CFR 415.440). BPT limitations are "no discharge of process waste water pollutants to navigable waters".

9.4.1.9 Chrome Pigments--

The manufacture of chrome pigments is covered by Subpart AH, Chrome Pigments Production Subcategory of the Inorganic Chemicals Manufacturing Point Source Category (40 CFR 415.340). BPT limitations are 0.14 kg/kkg of product (140 g/metric ton).

9.4.1.10 Television Picture Tubes--

Lead glass is used in fabricating television picture tubes, and aqueous lead discharges can result from the grinding and polishing of the tube envelopes. Lead discharges are controlled by Subpart K, Television Picture Tube Envelope Manufacturing Category of the Glass Manufacturing Point Source Category (40 CFR 426.110), which covers the process by which raw materials are melted in a furnace and processed into picture tube envelopes.

BPT limits on lead are 4.5 g/kg of furnace pull. BAT limits are lowered by a factor of 10, to 0.45 g/kkg of furnace pull.

9.4.1.11 Hand-Pressed and Blown Glass--

Subpart M, the Hand-Pressed and Blown Glass Manufacturing Subcategory of the Glass Manufacturing Point Source Category, covers the production of handpressed and blown lead glassware (40 CFR 426.130). There are no lead limitations for BPT. BAT imposes lead concentration limits of 0.1 mg/l in waste discharges.

9.4.1.12 Lead-Sheathed Rubber Hose--

The discharge of lead from the manufacture of lead-sheathed rubber hose is covered under Subpart G, the Large-Sized General Molded, Extruded and Fabricated Rubber Plants Subcategory of the Rubber Manufacturing Point Source Category (40 CFR 428.20). Both BPT and BAT lead limitations are 0.007 kg/kkg (0.7 g/metric ton) of raw material.

9.4.1.13 Gasoline Antiknock Additives--

The dispersal of lead to the atmosphere through the combustion of leaded gasoline is by far the largest loss of lead to the environment, dwarfing the total of all other emissions combined. Thus, it was appropriate that EPA regulations have been promulgated to reduce this major source of lead emissions.

The Environmental Protection Agency, on December 6, 1973, published (38 FR 33734) regulations designed to phase down the average lead content of gasoline by steps, down to 0.5 g/gal by January 1, 1979. The announcement stated that it was "the Administrator's judgement that the promulgated reduction schedule is reasonable from the standpoint of protection of health and from the standpoint of economic and technological feasibility".

In spite of this judgement the regulations were the subject of lengthy litigation until June, 1976, at which time the U.S. Supreme Court denied certiorari, effectively reinstating them. Due to the length of elapsed time, the phase down time schedule had to be modified somewhat. The date for compliance with the 0.5 g/gal level is now October 1, 1979. The January 1, 1978 standard of 0.8 g/gal remained in effect, but could be suspended if a refiner could show due dilignece incompliance efforts. Most large refiners were able to show due dilignece, and as a result the average lead content of the gasoline pool in 1978 was 1.20 g/gal (Table 4.16).

The small refiners (producing less then 25,000 barrels/day of gasoline) as facing proportionally higher costs to comply, with smaller financial resources to support the modifications. Accordingly, an October 1, 1979 level of 1.0 g/gal for small refiners was proposed by EPA (42 FR 3183, January 17, 1977). It was estimated that this would raise the ultimate lead average level to about 0.52 g/gal, with the effect on ambient lead concentrations considered to be negligible. However, the Congress included in the Clean Air Act Amendments of 1977 specific relief for the small refineries of small refiners. A new rule was promulgated by EPA in response to this amendment which provides for a sliding scale of lead content increasing to a maximum of 2.65 g/gal for a 5,000 barrels/day refinery (43 FR 17841-17842, April 26, 1978). It is estimated that this will affect about 5.5 percent of the U.S. gasoline supply and raise the average gasoline pool lead to 0.59 g/gal.

9.4.1.14 Lead-Based Paint--

On September 1, 1977 (42 FR 44192-44202) the Consumer Product Safety Commission (CPSC) issued regulations governing lead-containing paint and certain consumer products bearing such paint, with an effective date of February 28, 1978, which banned, as hazardous products:

(1) Lead-containing paint and similar surface-coating materials containing more than a safe level of lead.

(2) Toys and other articles intended for use by children bearing lead-containing paint or other similar surface-coating material containing more than a safe level of lead.

(3) Articles of furniture bearing lead-containing paint or other similar surface-coating materials containing more than a safe level of lead.

The question of what is a safe level of lead has gone through a series of revisions. At one time this was any paint containing less than 1 percent lead in the dried film. A later amendment to the Lead-Based Paint Poisoning Prevention Act (LBPPPA) reduced this to 0.5 percent, and directed the CPSC to determine if a level of lead in paint in excess of 0.06 percent but not over 0.5 percent was safe.

On February 16, 1977 (42 FR 9404), The Consumer Product Safety Commission announced its decision that available scientific information is insufficient to establish that a level of lead in paint above 0.06 percent but not over 0.5 percent is safe. The result of this decision was to classify any paint manufactured after June 22, 1977 containing more than 0.06 percent lead as lead-based paint, under the LBPPPA.

The regulations exempt major appliances such as refrigerators and ranges and household items such as venetian blinds from the "furniture article" category; and a subsequent amendment excludes metal furniture. Also exempted, if properly labelled were the following coatings:

(1) Agricultural, and industrial equipment refinish coatings.

(2) Industrial (and commercial building) maintenance coatings, including traffic and safety marking coatings.

(3) Graphic arts coatings (products marketed solely for application on billboards, road signs, and similar uses and for identification marking in industrial buildings).

(4) Touch-up coatings for automobiles, agricultural and industrial equipment, lawn and garden equipment, boats, outboard motors, motorized recreational vehicles, and appliances.

(5) Catalyzed coatings marketed sold solely for use on radio-controlled model powered air craft.

Exempted without the labeling requirement were:

(1) Lead-paint backed mirrors which are part of furniture articles.

(2) Artists paints and similar materials.

Excluded as not qualifying as consumer products were automotive and marine coatings.

9.4.1.15 Lead-Based Paint in Federally Owned or Assisted Housing--

The LBPPPA was first enacted in 1971 (P.L. 91-695) to deal with the problems of childhood lead poisoning caused by the ingestion of lead-based paints. The original act prohibited, after January 13, 1971, the use of lead-based paint in residential structures constructed or rehabilitated by the Federal government, or with Federal assistance in any form. The term "lead-based paint" was defined as any paint containing more than 1 percent lead in the dried paint film.

The LBPPPA was amended in 1973 (P.L. 93-151), which among other things redefined "lead-based paint" to mean any paint with over 0.5 percent lead in the dried film. The LBPPPA was again amended in 1976 by enactment of the National Consumer Health Information and Health Promotion Act (P.L. 94-317).

Pursuant to these various pieces of legislation, the Department of Housing and Urban Development (HUD) issued July 13, 1976 (41 FR 28876) regulations revising 24 CFR Part 35 governing numerous controls on HUD-associated housing constructed prior to 1950. These regulations, among other things:

(1) Require HUD to notify purchasers and tenants of HUD-associated housing constructed prior to 1950 of the hazards of lead-based paint poisoning.

(2) Prohibit the use of lead-based paint in HUD-associated housing.

(3) Provide for the elimination, to the extent practicable, of lead-based paint hazards in HUD-associated housing (and similarly in Federally-owned housing prior to sale for residential habitation).*

---

* "All surface conditions identified as immediate hazards are to be thoroughly cleaned (washed, sanded, wire-brushed, scraped or otherwise cleaned) as to remove all cracked, scaling, peeling, chipping, and loose paint on applicable surfaces. Surfaces shall then be repainted with two coats of a suitable non-leaded paint."

9.4.1.16 Occupational Exposure to Lead--

The Occupational Safety and Health Administration (OSHA) of the Department of Labor proposed (41 FR 45934, October 3, 1975) to amend 29 CFR Part 1910 to the effect that the permissible employee lead exposure limit to an 8-hour time-weighted average concentration, based on a 40-hour work week be 100 $\mu g/m^3$, rather than the present 200 $\mu g/m^3$ standard. The proposed regulations also governed the determination of employee exposure, methods of compliance, personal protective equipment and clothing, training, medical surveillance, and recordkeeping.

The final standard was promulgated on November 14, 1978 (43 FR 52952-53014), with an effective date of February 1, 1979. It differed from the proposed regulations in that the maximum allowable 8-hour time-weighted-average was reduced to 50 $\mu g/m^3$, with compliance dates by which this must be achieved varying from one year for "all other industries", to five years for lead pigment manufacturing, nonferrous foundries, battery manufacturing, and secondary lead production, and to ten years for primary lead production. The final regulations also govern protective equipment, and clothing, monitoring, housekeeping, hygiene facilities and practices, medical surveillance, medical removal protection, and employee training.

It is of interest to note that these regulations govern only occupational exposure of employees at their workplaces, and totally ignore emissions to the general environment outside the workplace. Thus, it is conceivable that the net result of the adoption of the 50 $\mu g/m^3$ limitation may well be to increase air lead emissions, since increasing ventilating air is one of the easiest and most economical means of reducing in-plant air lead concentrations.

9.4.1.17 Drinking Water Standard--

The maximum contaminant level for lead, applicable to community water systems (a public water system serving > 15 year-round service connections or > 25 year-round residents) is 0.050 mg/l (50 $\mu g/l$). (40 CFR 141.11).

9.4.1.18 Evaporated Milk--

On December 6, 1974, the Food and Drug Commission published proposed regulations limiting the lead content of evaporated milk and evaporated skim milk to 0.3 ppm (39 FR 42740). The limitation was proposed as a tolerance. "Limiting an unavoidable substance through use of a tolerance rather than an action level is appropriate when there are no changes foreseeable in the near future that might affect the appropriateness of the limitation established. The commissioner concludes that additional improvements in can technology, futher reducing the levels of lead in evaporated milk are not expected in the near future".

On February 18, 1978, the Acting Director of The Bureau of Foods, outlined a 5-year plan to reduce lead intake from soldered cans by 50 per cent as well as to further reduce the background levels in canned and other foods. It was indicated that an advance notice of proposed rulemaking would be issued in the fall of 1978, and it is expected that the question of lead in evaporated milk will be addressed in that document.

9.4.1.19 Ambient Air Standards--

The U.S. Environmental Protection Agency was mandated by the courts to develop an ambient air quality standard for lead, on the grounds that it had been judged a criteria pollutant. In response to this mandate a ambient air quality standard was issued (U.S. Environmental Protection Agency, 1978), which instituted a primary and secondary air lead standard of 1.5 $\mu g/m^3$, quarterly arithmetic mean.

As noted earlier, this ambient air quality standard, will indirectly govern many point source emissions.

9.4.1.20 Lead Arsenate--

The tolerances for residues of lead arsenate in or on raw agricultural commodities, as established by EPA (40 CFR 180.194) are as follows:

- 7 ppm of combined lead in or on apples, apricots, asparagus, blackberries, blueberries, boysenberries, celery, cherries, cranberries, currants, dewberries, eggplants, gooseberries, grapes, loganberries, mangoes, nectarines, peaches, pears, peppers, plums, quinces, raspberries, strawberries, tomatoes, and young berries

- 1 ppm of combined lead in or on citrus fruits.

In view of the importance which has been shown for lead ingested with food, this level is considered to be excessively high, and should be lowered. However, as of now, there appears to be negligible or nil lead arsenate being manufactured or used on food crops in the United States. Accordingly, no objections to a reduction (or possibly even a banning) would be anticipated, and the revision would be little more than a formality. Thus, such a limitation would fall into the insurance category, to preclude the recurrence of this lead exposure hazard at some possible future date.

9.4.1.21 Lead Shot--

On July 28, 1976, the Fish and Wildlife Service of the Department of the Interior published (41 FR 31395) a proposed regulation amending 50 CFR 20 to govern the use of lead shot in certain specific areas of the Atlantic Flyway. The amendment prohibits taking ducks, geese, and coots with a shotgun containing shells loaded with shot composed of any metal other than such material as may be determined to be non-toxic to migratory waterfowl. At the present time the only shot type available and approved is steel shot.

Over the past two seasons the FWS has required hunters along parts of the Atlantic and Mississippi flyways to switch from lead to non-toxic steel shot. By Fall, 1978, the regulations will apply to parts of the Central and Pacific flyways as well (Carter, 1977).

9.4.1.22 Ocean Dumping--

While the ocean dumping of lead-containing materials is directly controlled by the regulations embodied in 40 CFR 227, Criteria for the Evaluation of Permit Applications, the limitation is applicable in a roundabout way. Lead is named as one of the group of materials requiring special care, in the class of Strictly Regulated Dumping. This classification requires that evidence of the acceptability of proposed acts of disposal will be required from the applicant according to specified criteria. This will involve demonstration that the material proposed for disposal meets the limiting permissible concentration of total pollutants as defined, considering both the concentration of pollutants in the waste material itself, and the total mixing zone available for initial dilution and dispersion.

It is evident from the above that this limitation is applied on a case-by-case basis, which may be cumbersome at times, but which will not hamper its effectiveness.

9.4.2 Indirect Limitations

There are a number of indirect limitations applied to various industrial and manufacturing operations which to some extent also control the emission of lead to the environment. The two preeminent indirect controls are those on air borne particulates through New Source Performance Standards (NSPS), and on hydrocarbon vapor losses. Except for the alkyl leads, as in gasoline, lead will be present in particulate form, and control of particulates should also adequately control lead emissions. Data contained in the recent EPA report on control techniques for lead air emissions (U.S. Environmental Protection Agency, 1977c) indicate that excessive losses of lead are not occurring.

9.4.2.1 Primary Lead Smelters--

On January 15, 1976 (41 FR 2332) the U.S. Environmental Protection Agency promulgated New Source Performance Standards for Lead Smelters. Covering basically air emissions of particulates and sulfur dioxide (FR 40 60.180-.186). Maximum allowable particulate concentration is 50 μg/dscm (dry standard cubic meter), equivalent to 0.022 grains/dry standard cubic foot, and maximum allowable sulfur dioxide concentration was 0.065 percent by volume.

These two regulations also effectively control emissions of lead particulates. Compliance with the sulfur dioxide regulation essentially requires a sulfuric acid plant, the gaseous feed of which must be free of particulates to avoid plugging the contact catalyst bed, and this is a considerably more stringent requirement than the 50 mg/dscm.

9.4.2.2 Primary Copper Smelters--

On the same date analogous NSPS regulations were promulgated for primary copper smelters (40 CFR 60.160-.166). The same considerations apply here as to lead smelters. Also, identical limitations were promulgated at the same time for zinc smelters, but these are an insignificant source of lead.

9.4.2.3 Secondary Lead Smelters--

EPA regulations covering NSPS for secondary lead smelters were issued March 8, 1974 (39 FR 9308), with the same 50 mg/dscm (0.022 gr/dscf) limits as for primary smelters, but, of course, with no sulfur dioxide limitations. (40 CFR 60.120-.123).

9.4.2.4 Secondary Brass and Bronze Ingot Production Plants--

Regulations for secondary brass and bronze plants, another potential source of lead air emissions, were promulgated at the same time as those for secondary lead smelters, also with the 50 mg/dscm (40 CFR 60.130-.133).

9.4.2.5 Ferroalloy Production Facilities--

Regulations have been issued to limit emissions of particulates from ferroalloy electric submerged arc furnaces. Regulations are in terms of emissions per unit of energy input, and range from 0.23 kg/mw-hr for one group of alloys, to 0.45 kg/mw-hr for some of the silicon-based alloys (40 CFR 60.260-.266).

9.4.2.6 Electric Arc Steel Furnaces--

Standard of performance for particulate matter is that gases discharged from a control device shall not contain particulate matter in excess of 12 mg/dscm (0.0052 gr/dscf) (40 CFR 60.270-.275).

9.4.2.7 Incinerators--

NSPS limitations on incinerators (above 45 metric ton/day) have been established by 40 CFR 60.52, at 0.18 g/dscm (0.08 gr/dscf).

9.4.2.8 Sludge Incinerators--

40 CFR 60.152 sets NSPS limitations for particulates from sewage sludge incinerators at 0.65 g/kg dry sludge (1.30 lb/ton).

9.4.2.9 Portland Cement Plants--

By 40 CFR 60.62, standards for particulate matter are set at 0.15 kg/metric ton of feed (dry basis) to the kiln.

9.4.2.10 Petroleum Storage--

Emission of gasoline from storage vessels and hence of antiknock in leaded gasolines is limited by the 40 CFR 60.112 requirements that petroleum liquids with a true vapor pressure of over 1.5 psia, but not greater than 11.1 psia, be stored in vessels with floating roofs or with a vapor recovery system. This will include all gasolines.

9.4.2.11 Gasoline Vapor Recovery--

Gasoline vapor recovery regulations (Stage I) have been proposed by EPA, under Section 110(a) (1) of the Clean Air Act, via Transportation Control Plans to be incorporated into state implementation plans. As a part of the plan for sixteen air quality control regions (AQCR's), regulations were promulgated which required the recovery of 90 percent of the vapor (by weight) displaced during the filling of gasoline storage tanks.

Also proposed are analogous (Stage II) regulations governing the filling of vehicle gasoline tanks at the filling station. Instead of a percentage reduction, which would be difficult both to measure and to enforce, a mass loss is proposed. The final value has not yet been set, but is expected to be in the range of 0.4 to 0.6 g/gal.

The proposed standard also contains a limitation on the amount of spillage permitted. Spillage would be permitted to occur in up to 15 percent of all vehicle refuelings. This appears to be a rather unquantifiable and vague regulation and it is difficult to see how it will be applied.

9.4.3 Recent Enabling Legislation

Two acts have recently been passed by the U.S. Congress which have the potential of influencing the manufacture, use, and disposal of substances containing lead. The Toxic Substances Control Act (TSCA), P.L. 94-469 of October 11, 1976 grants the Administrator of the U.S. Environmental Protection Agency broad powers to regulate chemical substances and mixtures. Under Section 9 of that act, the Administrator is instructed to use the authorities of other Federal laws administered by EPA, unless it is in the public interest to use TSCA. The same philosophy applies to other Federal laws not administered by the Administrator, except that there is an inter-agency protocol to follow. In any event, the act establishes the possibility that lead could be regulated under TSCA, should this be deemed to be the most desirable course.

The Resource Conservation and Recovery Act (RCRA) of 1976 (P.L. 94-580) establishes requirements for the management of solid and hazardous wastes. Like its predecessor, the Solid Waste Disposal Act, RCRA will leave the promulgation of governing regulations and their enforcement to the individual states; there will not be specific Federal limitations. The Federal role is to consist of financial and technical assistance and leadership in the development, demonstration, and application of new and improved methods of waste management.

However, Federal criteria are to be established for identifying, transporting, treatment, and disposal of hazardous wastes; these are still in the draft stage and have not yet been promulgated. These criteria, when established, will very likely regulate how lead-bearing wastes are handled in landfills, and may provide criteria for their disposal of agricultural lands.

9.5 CASE STUDY: ANALYSIS OF ALTERNATIVES FOR LIMITATIONS ON LEAD INTAKE FROM FOODS AND BEVERAGES

At the conclusion of the first phase of this program, it was evident that the greatest opportunity for reducing the daily lead intake of the general population was via the ingestion route, specifically food and beverages. Further, the benefits of such a reduction would apply universally, and would include the two most sensitive segments of the population, children and pregnant women.

Since the funds available for the second phase of the program were limited, it was necessary to select one candidate limitation as a test case to develop the possible approaches to limiting lead intake, to determine the reductions which were attainable, their technological and economic feasibility, and to assess the benefits which would be conferred if adopted. Given these constraints, selection of adventitious lead in foods and beverages was an obvious choice for more detailed study, especially since the information available indicated that this problem was not under investigation elsewhere.

After the program was well under way it was learned that the Food and Drug Administration was engaged in a study of lead in foods with the objective of reducing lead contents, especially in the foods consumed by infants and young children. Thus, there may be some duplication between the two studies. However, it is hoped that the findings of this study will be useful in corroboration and support of the FDA efforts to reduce man's intake of lead from foods.

9.5.1 Lead in Processed Foods and Beverages

The lead concentrations in various foods, both processed and unprocessed, are described and discussed in Section 8.5.2. Processed foods never have less lead than the fresh food from which they were prepared, and often have more. Freezing, drying, or packaging in glass or aluminum contributes little or no lead to food. However the conventional three-piece lead-coldered can is still the standard for the food canning industry, although an evolutionary trend towards its replacement is evident, and this is a well-recognized source of lead in canned foods. Examples of the increase in lead content upon packing foods in cans with lead soldered side seams, and upon their continued storage were presented in Section 8.5.3. As illustrated by Table 8.20, doubling and tripling of the lead content was not unusual.

9.5.1.1 Food Can Technology--

Although soldered tin plate cans still dominate the processed food industry, changes are being made, both in the familiar three-piece soldered tinplate can as well as in other competing forming and joining techniques for manufacturing cans. Additionally, other packaging techniques, glass, plastic, retortable pouches (See Section 9.5.1.2) are competing with cans for the food market. In non-food items, other packages have often been extremely successful, e.g., fibre and composite cans have seized 77 percent of the 2.6 billion unit motor oil market (Kline & Company, 1977).

The demands of food canning are more rigorous than those for beverages; the reactions between foods and cans are more varied, and the need to prevent them has until now favored the three-piece soldered tin-lined can. For example, for many foods good substitutes for tin have not yet been commercially available. Also, tinplate does not lend itself to welding or cementing, although this can be accomplished by suitable edge treatment. However, this removes the protective tin coating at the seam and creates the problem of preventing contact of food and metal.

As other protective organic coatings are developed, and approved for food use by FDA, the other joining techniques will be better able to replace soldering. This trend seems to already be under way since it appears that the can manufacturers are no longer installing three-piece soldered can lines; new can lines are two-piece or use alternative joining technologies.

The other problem is the aseptic canning process. In simple terms, this involves packing food in open top cans, heating the cans to exhaust air from the food, sealing tops on the hot cans by a crimping operation, and then heating in a retort (customarily at about 240F) for a definite length of time adequate to destroy pathogenic organisms which might be present on the raw food material. Since the closure is made hot, upon cooling of the sterilized cans, the contraction of the contents produces a partial vacuum in the container.

The total and complete effectiveness of lead soldered cans has been demonstrated for many years; this is not so true for the newer processes. The rigors of the aseptic canning process also has prevented aluminum from enjoying much success. Aluminum, a higher cost metal, is competitive for beverages by the use of very thin gages, and now has a large share of the beverage market. However, the vacuum encountered during food canning is not compatible with thin-walled aluminum cans, and thicker gages are too costly to be competitive.

9.5.1.1.1 Lead-soldered cans--The lead-soldered food can has been used for several generations and to considerable degree has been responsible for the growth of the canned food industry. Canning of various products from the agricultural, fishing, cattle, and beverage industries is an important market outlet for these sections of the American economy. It is also an important market for packaging, labels, sanitation supplies, and for food and beverage processing machinery and equipment.

During recent years there has been a decline in the numbers of lead-soldered cans used in the food and beverage industries. A number of circumstances probably account for this including the advent of frozen food packaging a few decades ago, development of alternate methods of manufacturing cans, and more recently the desire to eliminate as much lead as possible from foods that are ingested by humans, especially small children.

Kopetz (1978) lists the following advantages for the soldered can:

Equipment exists
Well known technology
Essentially no new capital requirements
No size limitations
Good abuse resistance
Good integrity
Adequate shelf life
Easily used in customer's equipment

There has developed a variety of choices in can forming and joining alternatives. Materials have changed. The familiar tin plate has gradually grown to have much thinner coatings; and the former hot-dipped tin plate has been superseded by electrolytic tin plate; a less expensive substitute for tin plate, electrolytic chromium coated-steel has been developed and is designated TFS-CT (tin-free steel-chromium type) or TFS-CCO (tin-free steel-chromium-chromium oxide). This material can be used for many products where the cathodic protection normally supplied by tin is not needed. TFS is a primary material for cemented and welded beer and carbonated beverage containers, and can be used in sanitary food cans (Beese and Ludwigsen, 1974).

Aluminum, even though having a higher cost per pound than steel, is competitive where thin gages are practical. It has made its greatest penetration in the soft drink and beer can markets, where it claimed about 23 percent and 61 percent of 1976 shipments, respectively (Can Manufacturers Institute, 1977). Aluminum had 29 percent of the total 1976 can market, mostly due to its success in the beverage market.

Internal pressures are not high for beverages, and thin-walled aluminum cans will withstand them quite successfully. However, thin-walled aluminum cans will not withstand the pressures, and more specifically the vacuums of the aseptic canning necessary for foods; and using thicker walls increases the material cost out of the competitive range. A prohibition or severe limitations on lead solder in food cans might eliminate this cost barrier for aluminum food cans.

In addition to soldered-seams, joining techniques for three-piece cans include cemented and welded (forge-welded or wire-welded) seams; or two-piece cans can be formed by a drawing and redrawing process, or by the drawing and ironing process. The technology involved in each of these is described briefly in the following paragraphs, based in large part upon Kopetz's (1978) description. It should be remembered that these other processes are all possible alternatives to the lead-soldered can which have been reduced to practice and are in commercial use.

In the making of three-piece soldered cans the tin-coated plate is first sheared or slit into individual body blanks, which are fed into the bodymaker, where the blanks are notched, rolled into round cylindrical shapes, hooks formed on both edges, flux applied to these edges, and the edges hooked together to form the body of the can. In the next machine the cans, with seam side down, pass over a disc rotating in a bath of molten solder, which applies solder to the seam; the wetting action of the solder causes it to penetrate fully into the seam. After passing by a post heat burner, the excess solder is wiped from the outside of the can, and the can proceeds to a cooling section where the solder solidifies. The last operation may be the application of a stripe of lacquer to the soldered seam inside and outside.

The next operation on the can line is the flanger, which puts a flared rim on both ends of the can body. Finally, one end is double-seamed onto the can body. A coat of lacquer or enamel may be sprayed into the formed can body, in lieu of the stripe mentioned above. The cans are then tested under air pressure in equipment which automatically rejects any that have imperfect seams (Ellis, 1967).

The following advantages are recognized for the soldering process (Kopetz, 1978):

Equipment exists
Well known technology
Essentially no new capital equipment
No size limitations
Uses wide range of plate thicknesses and tempers
Good abuse resistance
Strong end profile
Adequate shelf life
Easily used in customers' equipment

9.5.1.1.2 Cemented cans--There are two types of cemented side seam cans. The first is similar to the soldered can, except that a cement has replaced the solder. It has been used in a number of non-thermally processed applications.

The other cemented container has a lap seam construction. The body blanks are preheated by direct flame and the adhesive transferred to the edge of the blank before its transfer to the bodymaker. After forming into cylinders and reheating the can is pressed together at the lap and chilled to set the adhesive. The remainder of the forming process is the same as for the soldered cans.

The technology has been used commercially for billions of beer and soft drink containers, and for millions of processed luncheon meat cans. However, the technology of this approach for sanitary food cans is still in the early development stages. The advantages of this process are taken to be:

Good can integrity
No solder, no flux
Capable of producing in all current sizes
Interchangeability with other three-piece cans
High speed production
Uses lower cost tin-free steel (TFS)
No changes to customer equipment
Good abuse resistance

The disadvantages are that the containers cannot stand aseptic canning, and the cements only work on enameled tin-free steel or aluminum surfaces. Therefore, products requiring a plain tinplate inner surface cannot be packed satisfactorily. Other problems involved in substituting cemented seams for soldered cans are equipment lacks for many sizes of cans, and the probable need to have baked inside spray coatings at temperatures close to the softening point of the adhesive. Also, tin-free steel lap seam cemented cans present problems of iron pickup by corrosive food products. Sulfide-bearing seafood and meat products which react with iron may therefore require aluminum. However, cemented seam cans should eventually reach the same level of efficiency as soldered cans (Kopetz, 1978).

9.5.1.1.3 Welded cans--Two types of welding processes (forge and wire) are used in the manufacture of cans. Forge welding produces a lap welded side seam on a container body. This process was developed for making containers from tin-free steel that has been chromium treated. Wire welding, which also produces a lap welded side seam, is used for tin plate. Both methods are in large scale commercial practice, forge welding for billions of beer and soft drink containers, and wire welding principally for household and industrial products. For sanitary food cans both techniques are in limited commercial use.

Edge preparation for forge welding is required to remove the oxide and chrome layers from about an 1/8-in. strip so that clean steel is available for welding. With adequate edge preparation black plate and tin plate can also be welded; protection of the welded area against corrosion is required.

The can forming process is analogous to that for soldered or cemented seam cans except that after forming the cylinder it is tack welded in a few places before passing between roller-type electrodes to produce the continuous welded seam. Thermoplastic or thermosetting powder resins are applied in a stripe along the seam while the metal is still hot and fused in place.

The wire welding process is based on the use of expendable wires which act as the electrical contact between the wheel-type electrodes and the can body. Otherwise the process is analogous to forge welding. For food containers an inside stripe would have to be applied to cover the weld area.

The advantages of these welding processes are (Kopetz, 1978):

Forge Welding	Wire Welding
Uses lower cost tin-free steel	Does not need edge preparation to weld tinplate
Good can integrity	Good can integrity

Forge Welding cont'd	Wire Welding cont'd
High speed production	High speed production
Tolerance to high processing temperatures	Tolerance to high processing temperatures
Elimination of solder, fluxes, cements	Elimination of solder, fluxes, cements
Less body material	Less body material
Improved and more reliable double seam	Relatively quick size change
Interchangeable with other 3-piece cans on customers equipment	Interchangeable with other 3-piece cans on customers equipment
Good dent resistance	Good dent resistance

The apparent disadvantages of the welding processes are that forge welding equipment is not available for all can diameters, but all diameters are available in wire welding. It would take several years to provide the can making equipment for all can sizes and products required. Time also would be required for development of economic coating materials.

9.5.1.1.4 Drawn and redrawn cans--Drawn cans are punched from flat sheet stock. Drawing requires the fewest number of operations from sheet stock to finished can and is considered a simple method. Cans can be made from steel, tin-free steel, tinplate, or aluminum by this process, which produces a two-piece can with no side seam. Shallow drawing can be done in a single operation; deeper drawing requires multi-stage draws. This is a slower operation than the three-piece can processes, resulting in a higher unit production cost.

The advantages claimed for the drawn cans are:

Superior can integrity
No solder or flux
No side seam or bottom end seam
Uses chromium treated tin-free steel, tinplate, or aluminum
Cans are stackable

9.5.1.1.5 Drawn and ironed cans--The D & I process is similar to the draw and redraw process except that the punch forces the formed cup down through successively smaller diameter dies, which thins the sidewall by stretching the metal. For example, a 0.130-in. sheet blank can winds up as a seamless can with an 0.0045-in. wall (Kaercher, 1972).

The can and dies are flooded with a mixture of coolant and lubricant during the ironing step, which must be thoroughly washed from the cans after the cans are completed. The variable height bodies must be trimmed to a uniform height, and it may be necessary to form circular beads in the side walls to supply rigidity. The advantages are essentially the same as those for drawn and redrawn cans.

The drawn and ironed process is widely used for aluminum beer cans; even in 1971 they had 10 percent of the total market (Kaercher, 1972).

Aluminum is easier to draw than steel, and rather thin-walled containers are compatible with beer packaging requirements.

The problems with both drawn and redrawn and D & I can technology for food canning are the difficulty in producing the wide diversity, often coupled with small volume, of the cans required by the food canning industry. Also, much of the can making industry's present equipment would become obsolete.

9.5.1.1.6 Non-lead soldered cans--It is possible to make soldered cans that are lead free. The solders used are all tin or tin-silver and appear to work satisfactorily. There are, however, disadvantages in depending on tin as a can material. First, only small quantities of tin ore concentrates are produced in the United States. The only tin smelter in this country, at Texas City, Texas, treats Bolivian tin concentrates. The major sources of tin metal are Malaysia, Thailand, Bolivia, and Indonesia. Reliance on imported raw materials and metal is always a dangerous situation because exports from the foreign countries can be cut off at any time for various reasons.

A major factor facing tinplate and can manufacturers is the scarcity and high price of tin. Dr. Leon Katz of American Can Company is quoted (Steinberg, 1977) as stating that tin may start losing its share of the food-can market if prices continue at current record levels. While there is no precise tin price level that will trigger a price move to tin-free steel and other packaging forms, can makers are thinking about alternatives to tin. Steinberg also quoted R. C. Stolk, vice president for procurement of American Can as stating that if tin gets to $6 a pound it could conceivably permit the substitution of other coatings. American Metal Market, June 15, 1978, quoted the market price of tin at $6.10 per pound and the Government Services Administration (GSA) sold stockpile tin for $5.68 per pound. The price of tin often vacillates depending on the supply and demand situation and actions of the International Tin Council.

Alloying silver with tin to make solder is also introducing a higher priced material. The American Metal Market quoted price for silver on June 15, 1978 was $5.29 per troy ounce or $76.96 per pound. About 66 percent of the domestic supply of silver is obtained as a byproduct of copper, lead, and zinc mining and refining. In 1976, just over 50 percent of the silver consumed in the United States was supplied by imports. Principal foreign suppliers are Canada, Mexico, Peru, and United Kingdom.

It appears that going to all tin or tin-silver solders and increasing the demand for these two metals at their high prices is not a feasible general alternative to lead-soldered cans. This approach may be feasible for special applications, and, in fact, tin solder is reported as being used by some manufacturers for infant foods (Sulek, 1977a).

9.5.1.2 Alternatives to Lead Soldered Food Cans--

The welded, cemented, drawn and redrawn, and drawn and ironed cans described above are all alternatives to lead soldered food cans, although still cans, and possessing all the strengths and weaknesses of cans. The substitution of tin or tin-silver solders is a possibility only for very limited use. There are indications that the can industry will be hard pressed to obtain the tin for tin plate, let alone tin for a high-tin solder.

There are also other non-can alternatives. Glass is historically the oldest, and has been used for many years. A very new package is the retortable pouch, which is described in 9.5.1.2.2. Frozen foods are classified as supplementary to canned foods, but not alternatives in the context of this analysis, which has the constraint that to serve the market served by canned food storage at room temperature must be possible.

9.5.1.2.1 Glass containers--Glass containers have been used for packaging various food items as well as beer, soft drinks and fruit juices. The glass container producers are optimistic that they will continue to gain a larger share of these markets. Makers of baby food say their fruit juice sales have increased markedly since they switched from metal cans to glass containers that can be used with the standard tops for nursing bottles (Starr, 1978). According to Shakin (1977) the glass bottle is capturing more business in beer and soft drink packaging and capacity has been expanded by the major producers. A major reason for the increasing use of glass containers is their price advantage over metal cans. Assuming that the prices of steel and aluminum will rise faster than the prices for glass, the glass container should continue to enlarge its share of the market. Shipments of glass containers in 1976 ran to $3.4 billion, up from $3 billion in 1975.

The replacement of soldered cans by glass containers in high temperature food processing is not envisioned because of the characteristics of glass with respect to thermal shock and to impact.

9.5.1.2.2 Retortable pouches--A recent development in food processing and packaging is the advent of the retortable pouch. The pouch is made of aluminum foil sandwiched between plastic sheets, and is basically a can with flexible sides. As described by Howard (1978), the pouches are filled with food on the canning line, the air evacuated, and the pouches sealed and cooked under pressure until harmful bacteria are killed. Because the pouch is long and thin, heat transfer is better, reducing processing time and minimizing canning's tendency to overcook food; it is claimed the food tastes as good as frozen food. The pouches can be stored at room temperature, and cooked by dropping the pouch into boiling water. The technique, originally developed by the military for canned combat rations, has been described as the greatest development in food processing since the tin can.

There are obstacles that must be overcome before the retortable pouch makes much of an inroad into the canned food market. The filling equipment is crude, expensive, and scarce. The filling lines are also slow compared to canning lines. The food companies are concerned whether the large investment they would have to make for pouched food lines could really pay off. Last, but not least, is whether consumers will buy food in pouches because it is foreign to present day methods of preparing and packaging food.

Breaking into the food packaging market on any reasonable level will be an uphill battle for the backers of retortable pouches.

9.5.1.3 Good Manufacturing Practices--

One alternative to changing solder compositions, or can making technology, is the rather simple one of improving manufacturing practices and quality control; although simple, it can provide some very real benefits in reducing the lead content of canned foods, as will be shown for some specific foods.

The good manufacturing practice (GMP) concept is used frequently by the Food and Drug Administration in regulating the industries which it oversees. While the term is difficult to define absolutely, its meaning is easily understood, and it has apparently been quite successful as a general catch-all control tool for regulating the manufacture of foods and drugs.

9.5.1.3.1 Evaporated milk--This approach appeared to in part be responsible for the reduction achieved in the lead content of evaporated milk over the 1972-1974 period. As described in the proposed 0.3 ppm tolerance for lead in evaporated milk and evaporated skim milk (U.S. Department of Health, Education, and Welfare, 1974), the FDA had conducted an investigation early in 1972 to determine baseline values in raw and evaporated milk. Raw milk ranged from none to 0.28 ppm (av. 0.09 ppm) and evaporated milk ranged from 0.21 to 1.10 ppm (av. 0.52 ppm). These results were supported by other investigations reported in the literature (raw milk 0.05 ppm average; evaporated milk 0.35 ppm average). Illustrative data of the decreasing lead concentrations are presented in Table 8.16.

The FDA informed the evaporated milk industry of the seriousness of the problem and the need to develop a solution. After some industry studies, some modification in can manufacturing procedured were made in an effort to reduce lead levels in evaporated milk. These efforts were fairly successful. Analysis of over 3,000 samples from the first half of 1973 showed an average of 0.12 ppm. Analysis of 3,015 samples from the second half of 1973 and the first four months of 1974 also confirmed the 0.12 ppm average (range 0.02 to 0.33 ppm). Refinements in analytical techniques for better sensitivity at trace levels led to a lower value for raw milk, 0.021 ppm, based on the analysis of 80 samples. While a larger number of samples would have been needed to verify this value, it was concluded that the average was significantly less than the 0.09 ppm initially reported.

The reduction in lead uptake has been maintained. Dr. H. R. Roberts, Acting Director of the Bureau of Foods of FDA, stated recently (Roberts, 1978) that the average lead content of evaporated milk is about 0.1 ppm.

9.5.1.3.2 Canned Foods--The increases in lead content in canned foods, as demonstrated by the Can Manufacturers Institute-National Canners Association (CMI-NCA) 1974 canned foods survey (Sulek, 1977), were reported in Section 8.4.5 (Table 8.20).

The canning industry has since then been modifying can making procedures in an effort to reduce lead levels. The concept of GMP in preventing or cleaning up lead solder spatter and dust from the interior of the cans was an important element in the modified procedures. Of key importance in this work was the knowledge that lead dust, as opposed to structural lead (lead used to solder the sideseam) was the primary contributor to comtainer-oriented lead pick-up. Lead dust is a by-product of the soldering operation and techniques have been found to greatly control this phase of the operation. Structural lead, on the other hand, has been found to contribute very little lead to most canned items except in very aggressive products.

To follow up on the practical effectiveness of these equipment modifications and improvements in manufacturing procedures, another survey was conducted in 1976 by The Can Manufactures Institute and The National Food Processors Association (formerly The National Canners Association). Prefacing the presentation of the results, it should be noted that most of these data were obtained from cans produced on lines which had been modified to significantly minimize lead contamination of the containers. Due to the time and expense involved in these modifications, not all can manufacturing lines have been modified to date (Sulek, 1978). However, it is representative of what can be accomplished and comparable values should be the norm after all lines have been upgraded.

Thirteen products, suggested by FDA as among those likely to be fed to young children, were surveyed. Results for these selected foods are presented in Table 9.4, along with comparable data from the 1974 CMI/NCA survey, for those foods, included in the earlier (pre-modification) survey. As shown in Table 9.4 the average lead content for all foods tested was 0.19 ppm, and no product exceeded 0.30 ppm. Twenty-five samples of each product were to be surveyed. The sampling plan involved taking a 2-lb. sample at the filler bowl, and then 24 cans of finished product, which were stored 6 months before analysis (only 6 months storage was used in this study as the previous 1974 study had indicated insignificant increases beyond that time). In order to ensure that a representative sample was analyzed, 12 cans were composited, and a 1-lb. subsample removed, which was then totally solubilized in nitric acid for analysis.

The effectiveness of the process modifications is best illustrated by comparing the 1976 results with those of the 1974 survey (Table 9.4). All foods except vegetable soup showed a significant drop in lead levels. The average reduction for the 10 foods for which a comparison could be made was slightly above 40 percent.

Based on these results, a 50 percent decrease in adventitious lead from canned foods should be quite possible, without any major changes in packaging or in present can technology. Such a reduction would appear to be highly desirable, on an interim basis at least, while the evolutionary changes in the can industry needed to essentially eliminate all lead pick-up are implemented.

TABLE 9.4 COMPARISON OF RESULTS OF 1974 AND 1976 CANNED FOOD SURVEYS[a]

	Lead content, ppm				
	Filler bowl		Canned Product[b]		Percent change in lead level
	1974	1976	1974	1976	
Applejuice	0.02	0.02	0.17	0.13	-24
Applesauce	0.08	0.01	0.32	0.20	-38
Beef stew	0.11	0.03	0.29	0.19	-34
Chicken noodle soup	0.25[d]	0.02	0.68	0.25	-63
Corn (whole kernel)	0.08	0.05	0.32	0.22	-31
Green beans	0.18	0.04	0.35	0.15	-57
Fruit drinks	-	0.03	0.13	0.11	-15
Orange juice	-	0.03	-	0.07[c]	-
Peas	0.08	0.03	0.40	0.23	-43
Peaches	-	0.07	-	0.21	-
Pork & beans	0.23[e]	0.03	0.50	0.17	-66
Spaghetti	-	0.05	-	0.11[c]	-
Vegetable soup	0.07	0.02	0.25	0.29	+16
Overall average	0.12	0.03	0.32	0.19	-41

---

[a] Source: 1976-1977 CMI/NFPA Lead in Canned Food Survey (Sulek, 1978).

[b] Samples stored for 6 months before analysis

[c] Not included in calculation of average since they were not surveyed in 1974.

[d] Based on only one sample.

[e] Based on three samples.

Interestingly, while the lead content of the canned foods decreased as a result of improved can making procedures, more accurate analyses resulted in lower average lead contents of the raw foods. Where the average lead content of samples from the filler bowl was 0.12 ppm in the 1974 survey this decreased to 0.03 in the 1976 survey. (During the entire survey a total of 2276 unprocessed samples were analyzed, for which the overall average was 0.06 ppm.) Taking the 0.03 ppm value, the raw food represents 16 percent of the total lead in these selected foods. Thus, according to the 1976 survey data, the adventitious lead, instead of comprising 2/3 of the total as formerly estimated, and as confirmed by the 1974 survey, now represents 84 percent of the total intake. However, this mathematical quirk should not obscure the fact that the absolute lead intake from canned food would be cut nearly in half at the new level.

9.5.1.3.3 Infant foods--Another food industry segment that has exhibited continuing reduction in lead content is infant food, under the sponsorship of the Infant Food Manufacturer's Lead Program. In order to reduce lead levels, manufacturers have converted to either tin solder or a double spray and bake can lining; both of these have increased can costs (Sulek, 1978). However, the benefits to the most sensitive segment of the general population have been significant, as illustrated by the analytical data contained in Table 9.5. In 1973 the mean lead content in canned infant juices was 0.29 ppm. In 1974, when new can making procedures were begun the mean lead level was reduced 52 percent to 0.14 ppm. The reductions have continued; in the first half of 1977 the level was down to 0.06, a reduction of nearly 80 percent from 1973, and approaching the "background" level.

9.5.1.4 Economic Effects of Limitations Alternatives--

The number of cans produced by the can industry is astronomical, over 84 billion in 1976. Food cans accounted for 29.4 billion, beverage cans 46.4 billion, pet food cans 3.1 billion, and general packaging cans (motor oil, paints, aerosol cans, and all others) for 5.5 billion (Can Manufacturers Institute, 1977). Of the 46.4 billion beverage cans, 19.5 billion were soft drink cans, and 26.9 billion were beer cans. Aluminum cans had 61 percent (16.5 billion) of the beer can market and about 23 percent (4.4 billion) of the soft drink can market. It is evident from these data that the can industry is characterized by a low-unit cost, very high-volume product, and that changes in manufacturing costs of a fraction of a cent per can are important.

Alternative processes for making cans not requiring a lead solder side seam are available, as described in Section 9.5.1.2. None are as well suited as sanitary food cans as the present 3-piece soldered can. One approach uses other joining processes-cementing or welding. In another, the cans are drawn, giving a 2-piece can with no side or bottom joint. A major disadvantage of these alternative techniques is their requirement for a very high volume of a single size to be economic. The food can industry is characterized by many sizes, with no one size possessing a dominant volume comparable, for example, to the standard 12-oz. beverage can. While some standardization of food can sizes is undoubtedly possible, it appears somewhat doubtful that the number could be reduced to a level suited to these alternative processes. As described in the following pages, these alternative

Table 9.5 LEAD CONTENT OF INFANT JUICES PACKED IN METAL CONTAINERS[a]

Infant Juice	Lead content, ppm 1973	1974	1975	1977[b]
Apple	0.36	0.14	0.05	0.03
Apple-cherry	0.28	0.18	0.11	0.03
Apple-grape	0.26	0.16	0.12	0.03
Apple-apricot	0.16	0.21	0.20	0.06
Apple-plum	-	-	-	0.03
Apple-pineapple	0.26	0.16	0.13	0.06
Apple-peach	-	-	0.04	0.06
Apple-prune	0.19	0.14	-	0.03
Mixed fruit	0.39	0.16	0.09	0.07
Orange	0.47	0.14	0.08	0.07
Orange-apple	0.25	0.08	-	0.08
Orange-apricot	0.27	0.14	-	0.10
Orange-apple-banana	0.54	0.17	0.18	0.09
Orange-banana	0.11	0.08	0.07	0.02
Orange-pineapple	0.21	0.15	0.06	0.07
Prune-orange	0.27	0.10	-	0.08
Number of samples	-	841	1138	675
Average ,ppm	0.29	0.14	0.10	0.06
Reduction from 1973 ,percent	-	52	66	79

[a] Source: 1976-1977 CMI/NFPA Lead in Canned Food Survey (Sulek, 1978).

[b] First half of 1977.

processes are characterized by high capital costs, and their substitution would require major expenditures by the can industry.

9.5.1.4.1 Industry structure and production--The manufacture of food cans is but one part, although the largest part, of the can making industry. According to Ellis (1967) cans then accounted for nearly one-third of all units of consumer packaging, and were used for some 2,500 different products. In more recent data Kopetz (1978) stated that the can makers had total sales of $8 billion in 1977, and that about 58 percent of the metal can sales were three-piece cans used by the food and beverage industries. The Bureau of Census reports that the total number of cans shipped in 1976 was 84 billion valued at $6.4 billion, up from a value of $5.9 billion in 1975. Employment in can making establishments is about 60,000 people working in 433 plants located in 277 different municipalities. The combined total payroll for 1976 was estimated at $1.2 billion. In addition, can makers have annual capital expenditures of $142 million.

The can industry is among the most highly concentrated in packaging. In 1975 two companies, American Can and Continental Can, accounted for 37 percent of the value of all production (down from 56 percent in 1970) and 50 percent of all merchant sales. National Can and Crown Cork & Seal accounted for an additional 18 percent of merchant sales. The top eleven companies, all with merchant can sales of $30 million or more, had 78 percent of the merchant total. These companies and their estimated sales of cans in 1975 are shown in Table 9.6.

Table 9.6 MAJOR U.S. MERCHANT PRODUCERS OF METAL CANS, 1975

Rank	Company	$ Million
1	Continental Group	$1,175
2	American Can	1,125
3	National Can	535
4	Crown Cork & Seal	315
5	Reynolds Metals	150
6	Ball Corp.	70
7	Diamond International	60
8	Van Dorn	60
9	Kaiser Alum. & Chemical	55
10	J.L. Clark	30
10	Sherwin-Williams	30
	Other merchant suppliers	1,020
	Captive producers	1,545
	TOTAL	$6,170

Source: Estimates by C.H. Kline & Co. (1977)

At least 30 firms make cans solely for their own use. They include such well-known food processors as Borden, California Canners & Growers, Campbell Soup, Carnation, Castle & Cooke, General Foods, Green Giant, H.J. Heinz, Hershey Foods, Pet, Stokely-Van Camp, and Libby, McNeil & Libby. Captive manufacturers for nonfood products include R. Reynolds, United States Tobacco, and Texaco (Kline & Company, 1977).

Captive production grew at a rate of about 15 percent a year between 1970 and 1974, and is continuing to increase. It represented about 17 percent of total production, and had grown to nearly 30 percent by 1975. During the same period merchant shipments rose only about 2.5 percent per year. Due to the desire of large packers to reduce package costs, this trend should continue (Kline & Company, 1977).

An example of this trend is the new Metal Container Corporation (subsidiary of Anheuser-Busch, Inc.) can making plant in Columbus, Ohio. This plant will produce 800 million steel drawn-and-ironed cans annually for the Columbus brewery. The plant operates four can lines 24 hours a day, 7 days a week, employs more than 250 hourly-rated workers and represents an investment of over $25 million. Metal Container also operates an aluminum can making plant in Jacksonville, Florida, to serve the Anheuser-Busch brewery there.

Beer cans are standard at a 12-oz. size, as are soft drinks. However, in food cans there is a very large number of sizes. For example, for three-piece cans there are 270 different can sizes, with 16 different can diameters* (Kopetz, 1978).

Food cans are a very important part of the U.S. food chain. In 1975, almost 47 billion pounds of food and beverage (about 14 percent of the 337 billion pound total) was packed in three-piece containers, and was valued at $27 billion. Food canning activity was carried out in over 1,700 food processing plants located in 47 states, American Samoa, and Puerto Rico. Employment in the food canning industry averages about 140,000 during the seasonal low production period (February) and exceeds 275,000 at the seasonal peak of canning activity in September. The combined payroll in this industry in 1975 was approximately $1.7 billion (Kopetz, 1978).

9.5.1.4.2 Costs to adopt alternative can manufacturing processes--Present sanitary food can manufacturing processes, and several leading candidate alternative can manufacturing processes were described in Section 9.5.2.1. Several of these alternative processes require an organic coating stripe along the seam, to protect the metal from the contents of the can. A 360-degree spray coating on the inside of some food cans is already used in some applications, primarily to prevent reactions between can and contents. These organic coatings could also be used for the purpose of preventing lead pickup in lead-soldered food cans, although this technique is evidently used now only for special situations, e.g., infant foods.

---

*The can industry specifies cans by a 3-digit numbering system of inches and sixteenths for diameter x height, e.g., a 303x406 can is 3 3/16-in dia x 4 3/8-in height.

The capital and incremental operating costs for nine alternative production techniques have been estimated by representatives of the Can Manufacturers Institute on the joint CMI/NFPA Committee on Lead In Canned Foods. Preliminary findings of this study are contained in a recent draft report (Can Manufacturers Institute, 1978). The following discussion is a condensation and summarization of this draft report. One of the alternatives, induction soldering, is not considered here, since this variation on present soldering technology appeared to have little effect on the problem of lead pickup.

The eight remaining alternatives considered, and a brief characterization of each are as follows:

(1) Line Modification and Clean-Up

This involves modifying certain equipment, primarily that at the solder station, and the introduction of more sophisticated housekeeping procedures and equipment, buttressed by rigorous quality control procedures. This is the basic approach followed in achieving the 40 percent reductions in lead content described in Section 9.5.1.3. Adoption of these measures is also a prerequisite for the following three alternatives which also retain the lead soldered side seam.

There are few technological problems with this approach, and costs to upgrade existing systems are low to moderate.

(2) Inside Side Seam Stripe Using Organic Coatings

A stripe of an organic coating is applied to the side seam post-soldering. Incorporation of alternative (1) is required to realize the optimum benefits from this process. Some cans are already being produced using this technique.

There are few technological problems with this approach, and costs to modify existing systems would be moderate. Organic coatings compatible with all products are not available, and further development will be required.

(3) Inside 360-Degree Spray Organic Coating

This alternative requires coating material to be sprayed uniformly on the inside of the can after the side seam has been soldered. Some cans are currently being manufactured by this process. Incorporation of alternative (1) is also required to realize the maximum benefit from this process.

There will be technological problems in perfecting coatings suitable for the majority of food products and in obtaining

FDA approval of their safety, and coatings development and testing costs will be significant. Costs to add a separate spray coating process operation to existing lines are estimated to be major.

(4) Combination Inside Side Seam Stripe and 360-Degree Spray Organic Coating

This alternative includes the combination of alternatives (2) and (3) for greater protection; again incorporation of alternative (1) is a prerequisite to realize maximum benefits.

Costs are slightly higher than for alternative (3).

(5) Pure Tin Solder

This alternative would require almost no change to existing lines, but would require more tin than is annually produced. The low melting point of tin would pose some problems in the high temperature presterilization of formed cans prior to aseptic canning.

(6) Cemented Side Seam

This alternative is used widely for beer and soft drink cans, but is not suitable in its present state of development for aseptic canning (See Section 9.5.1.1.2). Considerable development in cements, coatings, and in forming processes would be required. Twenty-five percent of current investment in food can lines would become obsolete if this alternative is adopted. Cost to convert would approach $1 million per line, and changeover is estimated to require at least a five- year period, depending on rapidity of solution of technological problems.

(7) Welded Side Seam

Two types of welding processes, forge and wire welding, are used in the manufacture of cans (See Section 9.5.1.1.3), both methods are in large scale use, e.g. for beer, soft drinks, and household products, but the techniques have to date had only limited application to sanitary food cans.

The replacement of existing can manufacturing equipment would be costly and loss of 25 to 50 percent of current in - place investment may result. A major technological problem would be the adoptation of this high-volume single-size can process to the smaller volume of a multiplicity of can sizes characteristic of sanitary food cans.

(8) Drawn and Redrawn/Drawn and Ironed

> Drawn and redrawn and drawn and ironed cans are described and discussed in Sections 9.5.1.1.4 and 9.5.1.1.5. This method of forming produces a two-piece can with no side seam, and hence no lead at all. Both of these processes are particularly suited to the production of massive volumes of one size of can, e.g. a 12-oz. beverage can. Much of the current can manufacturing equipment would become obsolete if this replacement were made; cost per line was estimated as in excess of $1 million. Current cost studies do not justify this type of equipment for sanitary food can manufacture.

Capital and incremental operating costs above present costs are summarized in Table 9.7. The costs are necessarily generalized; for example, they assume a line speed of 400 cans/minute, although line speeds vary in practice from 100 to 800 cans/minute, and assume an average of 30 million cans/line/year (actual rates vary from 20 to 60 million). Total industry costs are based on the existing approximately 1,085 lines in the U.S. producing sanitary food cans, and all costs are based on current dollars.

The pure tin solder alternative can be dismissed as a general solution for the food can manufacturing industry on the grounds of an insufficient supply; it may be appropriate for special problems such as canning infant foods and formulas. Of the remaining alternatives, the most cost-effective would appear to be the line modification and cleanup and/or the side seam stripe.

Total elimination of lead (options 6,7,8) would require new industry investment in the billion dollar range, and cause a significant disruption of the food can manufacturing industry. As pointed out by Dr. Howard R. Roberts, Acting Director, Bureau of Foods, in a speech before the National Food Processors Association (Roberts 1978b):

> Based on industry data, it is estimated that about 33 billion cans are used each year to package 10-15 percent of all food. Since alternative containers (glass, 2-piece cans, plastics) are not immediately available, eliminating the soldered 3-piece can overnight would mean unacceptable disruption of the food supply, especially with respect to fruits, vegetables, meat, and seafood. Further impacts of eliminating the soldered 3-piece can would be those on the over 140,000 permanent and 135,000 seasonal employees in these industries; the $3.7 billion spent on materials, and the $204 million spent on new equipment. But even neglecting this economic impact, eliminating the tin can would bring an unacceptable impact on the food supply and on the nutritional status of major segments of the population. Immediate elimination of the 3-piece can is thus not a viable alternative.

TABLE 9.7 SUMMARY OF ESTIMATED COST OF ALTERNATIVE FOOD CAN MANUFACTURING TECHNIQUES[a]

Alternative	Capital Cost,[b] Per Line	Capital Cost,[b] Industry Total, Million Dollars[c]	Added Operating Cost/1000 Cans
(1) Line modification and cleanup	$45,000	$ 48.8	$0.10 to 0.30
(2) Side seam stripe	18,500	20.1	0.20 to 0.45
Side seam stripe plus line modification and cleanup	63,500	68.9	0.30 to 0.75
(3) 360-Degree Spray	610,000 - 860,000	661.9 - 933.1	4.00 to 6.00
(4) Side seam stripe plus 360-Degree Spray	625,000 - 875,000	678.1 - 949.4	4.20 to 6.45
(5) Pure Tin Solder	5,000	5.4	5.00 to 7.00
(6) Cemented Side Seam	986,000	1,069.8	3.85[b]
(7) Welded Side Seam	880,000	954.8	3.10[b]
(8) Drawn and Redrawn	1,300,000	1,410.5	4.30[b]
Drawn and Ironed	2,500,000	2,712.5	--

[a] Source: Can Manufacturers Institute (1978)

[b] Cost reflects only depreciation on investment, other operating costs undetermined pending further development.

[c] Based on 1,085 sanitary food can lines in U.S.

9.5.1.4.3 Economic impacts upon materials suppliers--The major materials used in can manufacture are steel, tinplate, chromium treated steel, and aluminum. Tinplate and lead solder are the prime materials in the soldered cans. For the years 1970-1976 tinplate shipments averaged about 4.5 million metric tons annually, or about 5.5 percent of total steel shipments. The U.S. Bureau of Mines (1978) estimates that, in 1977, 31 percent or 16,000 metric tons of tin was used in cans and containers. The bulk of the tin was used in tinplate and a much smaller amount was in the solder.

Cans can be manufactured from tinplate by all of the newer can making processes--cemented, forged, or drawn. Some producers prefer to use chromium treated steel because it is cheaper. However, the elimination of the lead-soldered can would not affect steel shipments per se, because steel sheet will still be used for making cans by the other processes. Glass and aluminum are strong competitors to steel as containers for food and beverages. Capture of a greater share of this market by either or both of these materials would lower the amount of steel in containers.

Widespread acceptance of the retortable pouch would also restrict the quantities of steel, aluminum, and glass in food containers.

The loss of the market for the small quantities of tin in lead solder used for soldered cans should not adversely affect the tin industry.

The U.S. Bureau of Mines (Rathjen, 1978) estimates that 4,000 metric tons of lead were used in soldered food cans during 1976. The total lead consumption in cans and containers of all kinds was estimated at 20,000 metric tons. Total annual lead consumption in 1976 and 1977 was about 1.34 million metric tons. This includes both primary and secondary lead. The loss of a 4,000 metric ton market should have little impact on the lead industry, especially in relation to the much larger loss of lead for the gasoline anti knock market.

In summary, elimination of the lead-soldered can over a reasonable period of time should have little impact on most material suppliers. The quantities of lead and tin used in solder are reasonably small and the loss of this market would be minimal. The present competition between steel, aluminum, and glass as food and beverage containers will continue even if the soldered can is phased out. Suppliers of coating materials could see an expanding market as cans are manufactured by the newer processes.

9.5.1.5 Industry Trends and Outlook--

The conclusion drawn from the results of the analysis described in the preceding paragraphs is that a large fraction of the reduction sought in adventitious lead in canned food can be achieved without precipitously abandoning the three-piece lead soldered can, by relatively minor adjustments in can making technology and by adopting strict "good manufacturing practices". This might involve intensive rinsing or brushing and rinsing of the formed cans to remove all lead solder dust and spatter, or a double stripe over the soldered seam, or even a double spray and bake interior lining. From the viewpoint of least capital investment requirement and least disruption of the industry, while at the same time reducing adventitious lead down nearly to target levels, this approach has much to recommend it.

Some industry trends are already in motion, which will have a bearing on the lead problem. According to one industry observer (Menke, 1978), three-piece can lines are no longer being installed; all new can lines are two-piece, aluminum or steel. Such competitive pressures will help to accentuate the phasing out of three-piece soldered can lines. For example, Reynolds Metals Company, a large producer of aluminum cans, will soon be making steel cans in its new plant at Salisbury, North Carolina. The company says it will be able to switch from aluminum to steel cans "at a moment's notics". One objective is to develop data on comparative costs for the two kinds of cans. It is assumed that these are drawn cans (Starr, 1978),

The soft drink industry is perhaps most rapidly phasing out three-piece cans. There were a total of 19.5 billion soft drink cans in 1976 (Can Manufacturers Institute). According to Mr. Korab of the National Soft Drink Association, cans represented 34.5 percent of total soft drinks, and lead soldered cans were only 35 percent of that, or 12 percent of the total cans. (The rest were two-piece steel or aluminum, or three-piece cemented or welded seam cans.) The use of soldered cans is rapidly phasing down, and by 1980 only 0 to 5 percent of the cans (i.e., less than 2 percent of soft drinks) will be marketed in soldered cans (Korab, 1978). These are being replaced predominantly by aluminum cans.

Similarly, in beer cans the trend is away from three-piece cans to two-piece. Within the last year Anheuser-Busch has gone from three-piece steel cans to two-piece steel cans; the next move will likely be to aluminum (Abel, 1978). Three-piece beer can shipments decreased from 14.7 billion in 1972 to 6.6 billion in 1976, by which time they had only about 25 percent of the market (Can Manufacturer's Institute, 1977).

While possibly not classifiable as an industry trend, at least one infant formula manufacturer is featuring in advertisements that its formula "is now available in a new 14-oz. can that is free of lead solder. Eliminating solder reduces the infant's environmental exposure to lead." General adoption of this advertising approach could hasten the complete replacement of lead solder in infant foods.

Infant foods are probably a special case. For adult foods, the more probable action will be for the industry to adopt an evolutionary approach to minimize lead pickup from three-piece soldered cans while conducting the research and development necessary to achieve the ultimate lead-free goal.

9.5.2 Lead Glazed Foodware

A ceramic glaze is a thin glassy coating, generally containing silica, which is applied and fused onto a clayware body. After firing, during which the glaze becomes molten, the glaze closely resembles a glass. It is continuous, impervious, and almost completely insoluble when properly formulated, applied, and fired. Glazes are as varied as the various kinds of pottery and each kind is best with its appropriate glaze. Lead glazes are the most widespread in use and considered the best for all ordinary purposes. The glazes used by commercial dinnerware makers seal the surface of the dinnerware to make it shiny and very smooth, easier to clean, more resistant to wear, and less likely to hold germs and bits of foods. Thus, glazes not only have aesthetic reasons for their use, but some very important functional ones.

9.5.2.1 Glaze Technology--

Glazes are usually applied to unglazed ceramic ware by dipping, brushing, or spraying an aqueous slurry of powdered glaze constituents, after which the ware is fired at a temperature sufficient to melt and fuse the glaze. Glazes are multi-component mixtures, thus the possible variations are almost endless. Typical of the clear glazes used on hotel china is the following, which is contained in the ILZRO Manual, Lead Glazes for Dinnerware (International Lead Zinc Research Organization, Inc., 1974):

Base Glaze (mols)

0.066 K_2O		
0.179 Na_2O	0.340 Al_2O_3	
0.261 PbO	0.314 B_2O_3	3.669 SiO_2
0.494 CaO		

One of the purposes of the lead oxide is to serve as a flux, which reacts at high temperatures and helps dissolve all of the glaze components to form a glass. Lead oxide under these conditions is an active solvent, and in addition to its fluxing action, also lowers the glazing temperature.

9.5.3.1.1 Variables influencing lead release from glazed ceramic ware--

The composition of the glaze is one of the major variables affecting its durability, and with respect to lead, the ease and rate of lead solubility and release. Copper, for example, is known to significantly increase the lead solubility of a glaze, and is therefore, avoided in dinnerware. Strong cautions are expressed against its use, as for example, the caution, emphasized by italics, in a booklet for art potters and hobbyists (Lead Industries Association, Inc., 1972):

> Under no conditions should copper oxide be added to a lead glaze intended for food surfaces.

Through trial and error, and research conducted by the ceramics industry going back to the 1930's, glaze formulations have been developed, whose lead loss when properly fired is extremely low, a few tenths ppm.

Firing temperature is another very important variable. Temperatures in the ceramics industry are measured by Orton cones, which soften and sag over at different temperature ranges (at a specified standard heating rate), depending on the particular clay mixture used for that number cone. Cones range in number in descending order from 022 to 01 and then in ascending order from 1 to 36 with increasing temperature. The range of interest for ceramics is from No. 010 (equivalent temperature 894 C) to No. 12 (1326 C). Cones 3 - 5 (1168 C to 1196 C) are typical of production glazing temperatures.

The manual on glazes issued by the International Lead and Zinc Research Organization (ILZRO) stated that "tests made on a large number of Cone 3 - 5 clear lead glazes (commercial plant production samples) show lead releases values of less than 0.5 ppm, and in a number of cases, less than 0.1 ppm. Thus, the cone 3 - 5 glazes, which constitute the large part of all commercial dinnerware glazes used in this country, show very low lead releases, as has been found in a number of other laboratories during the past three decades." (ILZRO, 1974)

As reported by Eppler (1976), speaking at the 1974 International Conference on Ceramic Foodware Safety, "it has been shown conclusively that the typical cone 3 - 5 formulas used to glaze both vitreous and semivitreous dinnerware in the United States produce consistently low lead release values, in most cases less than 0.5 ppm and with low scatter. Although medical journals have reported some cases of lead poisoning resulting from the use of certain imported and hand-crafted dinnerware, we know of no case where the glaze allegedly responsible even approached the type of glaze used by major American dinnerware manufacturers."

Hobbyists, not having the kiln capabilities of manufacturers, tend to fire glazes in the 07 to 02 cone range (984 to 1120C), which is one of the reasons why hobbyist (and hand-crafted cottage-industry) ceramic ware has sometimes caused lead poisoning problems.

Another variable is glaze thickness. With thin application, a glaze will dissolve body constituents, which will change the composition and structure of the glaze, thereby making it more acid resistant. Conversely, with a heavier application (above 3 mils), the body constituents may not penetrate through to the glaze surface, and if the glaze is not of an inherently acid resistant type, higher lead release rates will result. Here again the commercial manufacturer has more control over glaze thickness (as well as the incentive to minimize glaze materials costs) than the hobbyist and excessive glaze application in commercial dinnerware is unlikely.

9.5.2.1.2 Lead release/glaze testing--Lead extractability is measured by an acid extraction test, in which the ware being tested is nearly filled with 4 percent acetic acid and allowed to stand at room temperature for 24 hours. Lead concentrations in the extractant above 7ppm (7 mg/l) are considered evidence of unacceptable extraction (based on average results for six units).

This method, originally published as FDA Laboratory Information Bulletin No. 834, is still the standard test in the United States. The ASTM has essentially the same test as ASTM C738-76 (American Society for Testing Materials, 1977). The ASTM Manual describes this as a severe test that is unlikely to be matched under the conditions of usage of such ceramic ware.

In 1974, a group of experts met in Geneva, Switzerland, under the auspices of the World Health Organization, to review testing methods, standards, and legislation and enforcement procedures. A second meeting, held in Geneva, in 1976, resulted in the development of slightly revised test procedures, but more importantly, some recommended new standards governing lead release (World Health Organization, 1976).

One of the key changes was the establishmentof new dinnerware categories, as summarized below:

Category	Characteristics	Allowable Lead, mg/l
Flatware	Internal depth below rim <25 mm	7.0
Holloware	Internal depth >25 mm	
Small	1.1 l or less	5.0
Large	1.1 l or more	2.5
Cookware	Glazed articles specifically intended for cooking	5.0*

---

*First 2-hours of 24-hour test at ∿ 100C

Subsequently the International Standards Organization (ISO/TC 166 SC1) circulated a slightly revised version (N14) of the one considered in 1976 to the appropriate recipients for a vote; voting is expected to be completed in about 9 months, with the expectation that the proposed tests and standards will be adopted (McLaren, 1978). The U.S. FDA is cognizant of these developments, and has had a representative at these meetings, and is now reviewing the draft proposal. It would appear that there is a fair chance that the FDA will adopt these new standards, or something very much like them.

9.5.2.2 Historical Perspective--

The involvement of the Food and Drug Administration in the testing of ceramic ware for extractable lead evidently began in 1970, as the result of an article in Good Housekeeping describing a lead poisoning incident from a piece of imported foreign ceramic ware. FDA considers lead from ceramic glazes an indirect food additive. Initially samples of ware from 10 to 12 countries were tested and numerous violations were discovered.

This program has continued, and from 2,000 to 3,000 samples per year may be tested (Steele, 1978). These are not intended to be representative samples, statistically selected, but are selected because the inspectors suspect they may have high release. Thus, the percentages violations are skewed, and are in no way representative of total imports. A major improvement has taken place since testing began in 1970. The 1970 - 1973 period violations were typically in the 10 - 11 percent range; now they are less than 5 percent. However, this does not tell the whole story. In the early days many leach solutions exceeded 1,000 ppm, and some were 2,000 to 10,000 ppm. Now, even those with release rates above the 7 ppm limit do not have these gross excesses. Whereas violations have occurred in a number of imported products, problems of any significance appear to be limited to a handful of foreign countries.

In 1971 FDA conducted a sampling program covering domestically produced glazed ceramic foodware. Since then the industry association, the U.S. Potters Association, has assumed the responsibility of conducting an extensive volunteer program with twice-a-year sampling; the results are forwarded to FDA. FDA periodically makes spot checks of the entire domestic industry. Since periodic testing has not recently indicated any problem with domestically produced dinnerware, it would indicate that the problem is correctable through good quality control, and is fully under control with respect to the hazard of extractable lead.

Although there have been a few well-publicized instances of lead poisoning arising from this cause, they are so rare as to be newsworthy enough to be made the subject of magazine articles. Also, if these articles are examined, it is seen that the ware falls into two classes: handcrafted, or imported foreign ware (possibly also handcrafted). In fact, at least in some instances they were souveniers of foreign travel, and would never be called upon to pass FDA inspection. Thus, the control of such ware may be beyond the reach of Federal regulatory agencies.

As far as the general public is concerned, the problem of excessive lead intake from improperly glazed dinnerware seems to be one of very low probability. The FDA domestic monitoring and the self-policing of the industry appears to be fully adequate to protect the public against even marginal lead exposure hazards from this source. Whereas continued import surveillance is required, the problem with imported dinnerware also seems to be well under control.

9.5.2.3 Alternatives to Lead Glazes--

Before considering alternatives to lead glazes it is worth summarizing the benefits that lead glazes confer, which would perhaps be lost by their abandonment. These have been well summarized by Eppler (1976):

(1) The strong fluxing action permits lower temperatures, and also increases flexibility in formulation to achieve low expansion and smooth surface.
(2) Lead reduces viscosity and widens allowable firing range.
(3) Lead produces low surface tension chiefly responsible for the flatness and high gloss; also contributes to ability of the glaze to heal over small surface imperfections.
(4) Imparts a high index of refraction which results in a brilliant appearance.
(5) Reduces tendency towards surface crystallization or devitrification.

This combination of desirable properties cannot yet entirely be reproduced on a production scale in the leadless type glazes.

There are leadless glazes, and much research has been conducted aimed at developing a satisfactory substitute for lead glazes. Many of these increased quantities of alkali and boron oxides to help provide the low fusion properties found in leaded glazes, but lose other properties in so doing, for example in effects on underglaze colors and crazing (Lead Industries Association, 1972). None appear to have been sufficiently successful to persuade the industry to switch from lead glazes.

One alternative would be to forego glazing entirely. However, apart from the aesthetic loss there are serious health objections. Unglazed pottery presents a different problem due to the fact that it can permit the growth of pathogenic bacteria which potentially may constitute a far more serious health hazard than that resulting from possible exposure to lead in the glaze. Thus, the glazing of pottery does in practice confer a beneficial health effect in terms of better hygiene.

9.5.2.3.1 Economic implications--If the present mode of control is continued, either with the present limitations on extractable lead, or the proposed ISO standards, there will be no major impacts upon the industry, although there may be limited economic consequences for certain manufacturers. Present glaze formulations and processing procedures, combined with adequate quality control, appears to already be fulfilling the objective of protecting the public health, and no changes are needed.

Should the abandonment of lead glazes be required, the industry disruption would be large. The new lead-free products would be more expensive, and probably inferior with respect to durability. There are, however, no indications that such drastic action is necessary or desirable.

9.5.3 Miscellaneous Sources of Ingested Lead

There are several miscellaneous potential sources of ingested lead which need to be briefly discussed. Each of them has but a negligible effect on the absorbed lead dose of the general population, and appears to be important only in exceptional and unusual situations. Data on the actual degree of risk are scant, and judgements are necessarily based largely on conjecture.

9.5.3.1 Decal-Decorated Glass Tumblers--

In July of 1977, the Massachusetts Department of Public Health announced that they had analyzed the exterior portion of a promotional glass tumbler decorated with a lead glaze decal and were able to extract some lead. An interagency task force (FDA, EPA, and CPSC) was formed to examine the issue and decide upon an appropriate course of action.

The principal findings of the Interagency task force can be summarized as follows (Hill, et al., 1978):

(1) The industry and the involved agencies agreed that under certain conditions, lead and cadmium can be extracted from decorated glass tumblers and that excessive levels can pose undesirable health risks.

(2) Most glasses tested were well within acceptable levels.

(3) The glassware industry expressed a willingness for a voluntary standard and adequate quality control.

(4) The task force concluded that the proposed positive steps are likely to be adequate to reduce whatever risk may be present, therefore, no formal rule-making is required at this time.

9.5.3.2 Lead Glass--

The situation with lead glass dinnerware in part resembles that of glazed ceramics and in part that of antique pewter. Lead is added to glass to give it a high index of refraction, which when decorated by the cutting of grooves, provides a brilliant appearance, much fancied for such items as decorative fruit bowls. Some stemware is also manufactured from lead glass. Characteristically, such lead glassware is expensive, which tends to limit its use. Lead glassware is not customarily used as everyday dinnerware, in this respect resembling antique lead pewter.

Like lead glazed ceramic ware, lead glass is subject to leaching if the ware was not properly manufactured. Some foreign decanters have shown up to 50 ppm by the standard acetic acid leach test (Ott, 1978). As in the glazed dinnerware, the problem appears to be one of quality control. West German and U.S. lead glassware has no problem in passing the test, and the problem is only with one or more other countries. The present FDA inspection program appears to be adequately coping with this limited problem, and no need for additional limitations is evident at this time.

9.5.3.3 Solder-Joined Food Vessels--

One source of ingested lead, whose extent was not determined in this investigation, is from the lead solder which has, in the past at least, been used to join assemblies of metallic vessels of various sorts which are used to contain or to prepare foods or beverages, e.g. electric coffee pots.

A recent article by Wigle and Charlebois (1978) described a clinical lead poisoning case of an infant in Toronto, Canada, which was traced to the solder used to seal the heating element into the base of the electric kettle used to prepare the infant's formula. A subsequent survey of 574 households in Ottawa showed that 42.5 percent of water samples boiled in this type of electric kettle exceeded the 50 ppb WHO (and U.S.) standard, and 9 percent exceeded 110 ppb. However, the conclusion of the investigation was that lead exposure from electric kettles may be a significant problem only in infants receiving formula prepared with boiled water.

As indicated above it is not known how serious the problem is in the U.S. It is known that at least a small number of industrial applications have substituted a tin solder (described as a 95% tin-5% silver composition) for lead-containing solders in coffee pots, kitchen and bathroom appliances, and ice-making equipment (The Silver Institute, 1975). The technical behavior of these nonlead solders is reported as equivalent or superior in terms of lower melting point, high-temperature strength, good wetting, and better hygienic properties.

This problem, if it is one, needs to be better defined before recommendations can be made.

9.5.3.4 Pewter Ware--

Pewter is the historical name for an alloy of tin and lead, the lead serving to harden the tin. High quality pewter had relatively low percentages of lead, because of the known ill effects from lead on humans. From about the twelfth century the trade in pewter in all Europear countries appears to have been well organized and by the fourteenth century pewterers were everywhere bound by statutes and edicts designed to prevent the ruin of their business by unscrupulous competitors (Hedges, 1960). However, lead was then, as now, so much cheaper than tin that the alloys were debased by excessive lead. This became a particular problem after the introduction of better glazes for dinnerware led to the displacement of pewter tableware. Some debased pewters contained as much as 40 to 50 percent lead.

There was a lead health hazard from the use of pewter, particularly that with excessive lead content. However, this antique pewter now occupies a predominantly decorative function, which has effectively eliminated it as a health hazard.

In the middle of the eighteenth century Britannia Metal was invented. This is an alloy of 90-95 percent tin with 4-8 percent antimony, and 1-2 percent copper, and should contain no more lead than is justifiable as an impurity. The best grades contain no lead. Modern pewter is Britannia Metal.

The tin accounts for its soft sheen, ductility, and resistance to corrosion. The antimony serves to whiten the metal and impart a hardness unknown to old pewter. The copper adds ductility and desirable working properties, but if present in more than a small percentage, adds an undesirable yellow color. Utensils made from modern pewter (Britannia Metal) have a high resistance to the action of almost all acids. Foods and drink may be served in them without fear of chemical action (Osburne and Wilber, 1938).

Summarily, modern pewter offers a minimum of lead exposure hazard, and the exposure of the general public to antique pewter vessels appears so unlikely that it can be neglected.

9.5.3.5 Drinking Water--

As discussed in Section 5.3.2, the general public, with few exceptions, is not exposed to drinking water containing more than the 50 ppm limit of the interim primary drinking water standard. The exceptions are in areas of soft, low pH water; even here, the public water supplies are well below the 50 ppm limit. The problem arises from the use of lead service pipe between the distribution main and the house, and the size of the problem is a function of the length of lead pipe for each individual case. The areas concerned are primarily in New England, where the water is soft and many houses are old enough to have had lead service pipes installed, with apparently some similar problems in the Pacific Northwest.

There are two alternatives. The certain and absolute cure is to remove the offending pipe and replace it with non-lead pipe. The second would necessitate that water distributed throughout the city be treated to make it non-corrosive to lead pipe. Increasing the pH to a substantially alkaline level (pH 9-10), and possible also adding a little carbonate hardness to the water seems, on the basis of evidence from EPA Region I, to offer a viable solution.

Since this is a rather unique situation, local in its effect, it is not regarded as a strong candidate for national limitations; it can probably best be handled as a local problem.

9.6 REFERENCES

Abel, J. 1978. Anheuser-Busch, Inc., Columbus, Ohio. Personal Communication to R. A. Ewing.

American Society for Testing and Materials. 1977. Standard Test Method for Lead and Cadmium Extracted From Glazed Ceramic Surfaces (ASTM C738-76).

Beese, R. E. and R. J. Ludwigsen. 1974. Trends in the Design of Food Containers. Advances in Chemistry Series, Number 135. Chemistry of Food Packaging. Amer. Chem. Soc.

Campbell, W. J. 1976. Metal In the Wastes We Burn? Env. Sci. Technol. 10(5):436-439.

Can Manufacturers Institute. 1977. Metal Cans Shipments Report 1976. Washington, D. C. 8 pp.

Can Manufacturers Institute. 1978. The Economic Impact of Alternative Measures for The Reduction of Lead Content in Sanitary cans. Draft Report. Washington, D. C. April. 19 pp.

Carter, L. J. 1977. Waterfowl Hunters Must Give Up Lead Shot. Science. 198:1232-1233.

Chisolm, J. J. 1977. Is Lead Poisoning Still a Problem? Clin. Chem. 23(2): 252-255.

Consumer Product Safety Commission. 1977. Lead Containing Paint. Banned Hazardous Products. 42 FR 44192-44202. Washington, D. C. March 2.

Ellis, R. F. 1967. Metal Containers for Food. In: Fundamentals of Food Processing Operations, J. L. Heid and M. A. Joslyn (Eds.) The AVI Publishing Company, Westport, Connecticut. 615-640 pp.

Eppler, R. A. 1976. Formulation and Processing of Ceramic Glazes for Low Lead Release. In: Proceedings of International Conference of Ceramic Foodware Safety, Geneva, Switzerland, November 12-14, 1974. J. F. Smith and M. G. McLaren (Eds.). Lead Industries Association, Inc. New York, N. Y. 252 pp.

Feldman, R. G. 1978. Urban Lead Mining: Lead Intoxication Among Deleaders. New England J. Med. 298(20):1143-1145.

Fine, P. R., C. W. Thomas, R. H. Suho, R. E. Cohnberg, and B. S. Flashner. 1972. Pediatric Blood Levels. A Study in 14 Illinois Cities of Intermediate Population. J. Amer. Med. Assoc. 221:1475-1479.

Gilsinn, J. F. 1972. Estimates of the Nature and Extent of Lead Paint Poisoning in the United States. U. S. Department of Commerce. National Bureau of Standards. Technical Note 746. December 1972. Washington, D. C.

Hammond, P. B. 1977. Human Health Implications. In: Lead in the Environment. A Report Prepared for the National Science Foundation W. R. Boggess (Ed.) U. S. Government Printing Office, Washington, D. C. 272 pp.

Hammond, P. B. 1978. Environmental Assessment. In: Review of the Environmental effects of Pollutants: VII. Lead. EPA-600/1-78-029. U. S. Environmental Protection Agency, Health Effects Research Laboratory, Cincinnati, Ohio. Various pagination.

Hedges, E. S. 1960. Tin and Its Alloys. Edward Arnold Publishers, Ltd. London,England. 16-23 pp.

Hile, J. P., D. Schmelzer, and W. Muir. 1978. Lead and Cadmium in Decorated Glass Tumblers - Interagency Task Force Report. Washington, D. C. November 13. 7 pp. and attachments.

Howard, N. 1978. Will the Retortable Pouch Pay Off? Dun's Review 111(2):72-74. February.

International Lead Zinc Research Organization, Inc. 1974. ILZRO Manual. Lead Glazes for Dinnerware. New York, N.Y. 171 pp.

International Standards Organization. 1977. Ceramic Ware in Contact with Food/Release of Toxic Materials--Methods of Test and Permissible Limits. ISO/TC 166. SC 1:N14. October 19.

Kaercher, R. W. 1972. Drawn-and-ironed Steel Cans. Modern Packaging. October, 1972. 66-70 pp.

Kline & Company, C. H. 1977. Marketing Guide to the Packaging Industry. P. Noble (ed.). Fairfield, New Jersey.

Kopetz, A. A. 1978. The Three-Piece Soldered Can and Alternate Containers. Presented at the National Food Processors Association Annual Convention, Chicago, Illinois. 22 pp.

Korab, R. C. 1978. National Soft Drink Association, Washington, D. C. Personal communication to R. A. Ewing.

Lead Industries Association, Inc. 1972. Facts About Lead Glazes for Art Potters and Hobbyists. New York, N.Y. 12 pp.

Lutz, G. A., A. R. Levin, S. G. Bloom, K. L. Nielsen, J. L. Cross, and D. L. Morrison. 1970. Technical Intelligence, and Project Information System for the Environmental Health Service, Vol. III. Lead Model Case Study, by Battelle Memorial Institute, Columbus, Ohio, for U. S. Department of Health, Education, and Welfare Environmental Health Service.

Mirick, W. and L. J. Nowacki. 1977. The Development of a Lead-Based Paint Hazard Abatement Utilizing Heat-Shrink Films. Report to Department of Housing and Urban Development. Battelle - Columbus, December 29. 44 pp.

McLaren, M. G. 1978. Chairman, Ceramics Department, Rutgers University, New Brunswick, New Jersey. Personal communication to R. A. Ewing.

Menke, P. 1978. Reynolds Metal Company, Richmond, Virginia. Personal communication to R. A. Ewing.

National Soft Drink Association. 1977. NSDA 1976 Sales Survey of the Soft Drink Industry. Washington, D. C. 16 pp.

Osburne, B. N., and G. O. Wilber. 1938. Pewter. International Textbook Co., Scranton, Pennsylvania. 17-19 pp.

Ott, R. 1978. Ceramics Department, Rutgers University, New Brunswick, New Jersey. Personal communication to R. A. Ewing.

Rathjen, J. 1978. U. S. Bureau of Mines, Washington, D. C. Personal communication to R. W. Hale.

Roberts, H. R. 1978a. Statement Before the Environmental Protection Agency Public Hearing on Proposed National Ambient Air Quality Standard-Lead. February 15.

Roberts, H. R. 1978b. What the Food Industry Can Expect from the FDA in the Future. Presented at the National Food Processors Association Annual Convention, Chicago, Illinois, February 13.

Sachs, H. K., L. A. Blanksma, E. F. Murray, and M. J. O'Connell, 1970. Ambulatory Treatment of Lead Poisoning: Report of 1,155 Cases. Pediatrics 46(3):389-396.

Shakin, B. 1977. Denting the Can? Glass Bottle Makers Capture More of the Market. Barron's. August 9. 5 pp.

Starr, H. G. 1978. Container Changeovers Reverberate in CPI. Chemical Week. 122(23):15 June 7.

Steele, E. 1978. Compliance Division, Food and Drug Administration, Washington, D. C. Personal communication to R. A. Ewing.

Steinberg, H. 1977. Rising Tin Prices Peril Markets: Canners Explore Alternative Materials. Amer. Metal Market. 85(218) November 9.

Sulek, A. 1977a. CMI-NCA 1974 Lead in Canned Food Survey. Unpublished Paper. National Food Processors Association, Washington, D. C. 10 pp.

Sulek, A. 1977b. CMI-NCA 1976 Lead in Raw Product Survey. Unpublished Paper. National Food Processors Association, Washington, D. C. 8 pp.

Sulek, A. 1978. The Evaluation of Lead in Raw and Canned Food. Unpublished Paper. National Food Processors Association. Washington, D. C. 11 pp.

Tepper, L. D. and L. S. Levin. 1975. A Survey of Air and Population Lead Levels in Selected American Communities. In: Lead. T. B. Griffin and J. H. Kuelson (Eds.) Environmental Quality and Safety, Supplement Vol. II. Georg Thieme Publishers, Stuttgart. 152-196 pp.

U. S. Bureau of Mines. 1978. Mineral Commodity Summaries, 1978. U. S. Department of the Interior. Washington, D. C.

U. S. Department of Health, Education, and Welfare. 1974. Lead in Evaporated Milk and Evaporated Skim Milk. Proposed Tolerance. Food and Drug Administration, Washington, D. C. 39 FR 42740-42748. December 4.

U. S. Department of Labor. 1978a. Occupational Exposure to Lead: Final Standard. Occupational Safety and Health Administration, Washington, D. C. 43 FR 52952-53014. November 14.

U. S. Department of Labor. 1978b. Occupational Exposure to Lead. Attachments to the Preamble for the Final Standard. Occupational Safety and Health Administration, Washington, D. C. 43 FR 54354-54509. November 21.

U. S. Environmental Protection Agency. 1977a. Air Quality Criteria for Lead. U. S. Environmental Protection Agency. EPA-600/3-77-017. Office of Research and Development, Washington, D. C. December. Various pagination.

U. S. Environmental Protection Agency. 1977b. Control Techniques for Lead Air Emissions. 2 Vols. EPA-450/2-77-012. Office of Air Quality Planning and Standards Research Triangle Park, North Carolina. December. Various pagination.

U. S. Environmental Protection Agency. 1978a. National Primary and Secondary Air Quality Standard for Lead. 43 FR 46246-46263. October 5.

U. S. Environmental Protection Agency. 1978b. Implementation Plans for Lead National Ambient Air Quality Standard. 43 FR 46264-46271. October 5.

Wigle, D. T., and E. J. Charlebois. 1978. Electric Kettles as a Source of Human Lead Exposure. Arch. Env. Health. 72-78 pp.

World Health Organization. 1976. Ceramic Foodware Safety, Sampling, Analysis, and Limits for Lead and Cadmium Release. Report of Meeting. WHO/Food Add/77.44.

APPENDIX A

TABLE A-1. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES AND FEMALES UNDER 1-YEAR OLD

Food Class and Substance	Normal Daily Lead Intake, ug	Lead Intake, ug/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	10.82	10.82	10.82
Milk, processed, canned	5.69	2.845	1.897
Milk, processed, other	9.35	9.35	9.35
Cream	1.22	1.22	1.22
Cheese and mixtures	0.12	0.12	0.12
Eggs	2.04	2.04	2.04
MEATS			
Beef, canned	0.06	0.03	0.02
Beef, other	1.38	1.38	1.38
Pork, canned	0.02	0.01	0.0067
Pork, other	0.57	0.57	0.57
Poultry	0.381	0.381	0.381
Fish, canned	0.0	0.0	0.0
Fish, other	0.0	0.0	0.0
Other and mixtures, canned	1.23	0.615	0.41
Other and mixtures, other	5.86	5.86	5.86
NUTS AND OILS			
Legumes and nuts	3.38	3.38	3.38
Fats and oils	0.03	0.03	0.03
BREADS			
Bread	0.42	0.42	0.42
Other baked goods	0.40	0.40	0.40
Cereals, pastes	5.136	5.136	5.136
SUGAR PRODUCTS			
Sugar	0.031	0.031	0.031
Candy, jam, etc.	0.63	0.63	0.63
BEVERAGES			
Tea	0.4	0.4	0.4
Coffee	0.0	0.0	0.0
Cola, canned	0.02	0.01	0.0067
Cola, other	0.18	0.18	0.18
Fruit drinks, canned	0.01	0.005	0.003
Fruit drinks, other	0.02	0.02	0.02
Other soft drinks, canned	0.001	0.0005	0.0003
Other soft drinks, other	0.02	0.02	0.02
Beer, canned	0.0	0.0	0.0
Beer, other	0.0	0.0	0.0
Wine	0.0	0.0	0.0
Other alcoholic drinks	0.0	0.0	0.0
TOMATOES AND VEGETABLES			
Tomatoes, canned	0.16	0.08	0.0533
Tomatoes, other	0.04	0.04	0.04
Tomato juice, canned	0.07	0.035	0.0233
Citrus, canned	0.02	0.01	0.0067
Citrus, other	0.52	0.52	0.52
Citrus juice, canned	0.59	0.295	0.197
Citrus juice, other	0.09	0.09	0.09
Dark green vegetables, canned	0.14	0.07	0.047
Dark green vegetables, other	0.08	0.08	0.08
Deep yellow vegetables, canned	0.57	0.285	0.19
Deep yellow vegetables, other	0.96	0.96	0.96
POTATOES AND FRUITS			
Potatoes, canned	0.01	0.005	0.003
Potatoes, other	0.30	0.30	0.30
Other vegetables, canned	6.85	3.425	2.833
Other vegetables, other	3.85	3.85	3.85
Other vegetable juice, canned	0.16	0.08	0.053
Other fruits, canned	7.61	3.805	2.537
Other fruits, other	3.27	3.27	3.27
Other fruit juice, canned	0.68	0.34	0.227
Other fruit juice, other	0.03	0.03	0.03
TOTAL	75.419	63.4735	60.042

TABLE A-2. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES AND FEMALES 1-2 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, μg	Lead Intake, μg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	9.20	9.20	9.20
Milk, processed, canned	4.84	2.42	1.613
Milk, processed, other	7.95	7.95	7.95
Cream	1.56	1.56	1.56
Cheese and mixtures	0.60	0.60	0.60
Eggs	4.87	4.87	4.87
MEATS			
Beef, canned	0.20	0.1	0.007
Beef, other	4.75	4.75	4.75
Pork, canned	0.15	0.075	0.05
Pork, other	4.023	4.023	4.023
Poultry	1.4	1.4	1.4
Fish, canned	0.35	0.175	0.117
Fish, other	1.16	1.16	1.16
Other and mixtures, canned	1.35	0.675	0.45
Other and mixtures, other	6.57	6.57	6.57
NUTS AND OILS			
Legumes and nuts	4.16	4.16	4.16
Fats and oils	0.156	0.156	0.156
BREADS			
Bread	3.276	3.276	3.276
Other baked goods	2.9	2.9	2.9
Cereals, pastes	8.03	8.03	8.03
SUGAR PRODUCTS			
Sugar	0.25	0.25	0.25
Candy, jam, etc.	1.54	1.54	1.54
BEVERAGES			
Tea	3.6	3.6	3.6
Coffee	0.14	0.14	0.14
Cola, canned	0.32	0.16	0.107
Cola, other	3.1	3.1	3.1
Fruit drinks, canned	0.16	0.08	0.053
Fruit drinks, other	0.35	0.35	0.35
Other soft drinks, canned	0.02	0.01	0.007
Other soft drinks, other	0.28	0.28	0.28
Beer, canned	0.0	0.0	0.0
Beer, other	0.0	0.0	0.0
Wine	0.0	0.0	0.0
Other alcoholic drinks	0.0	0.0	0.0
TOMATOES AND VEGETABLES			
Tomatoes, canned	1.92	0.96	0.64
Tomatoes, other	0.54	0.54	0.54
Tomato juice, canned	0.87	0.435	0.29
Citrus, canned	0.04	0.02	0.013
Citrus, other	1.14	1.14	1.14
Citrus juice, canned	1.29	0.645	0.43
Citrus juice, other	0.19	0.19	0.19
Dark green vegetables, canned	0.27	0.135	0.09
Dark green vegetables, other	0.17	0.17	0.17
Deep yellow vegetables, canned	0.29	0.145	0.097
Deep yellow vegetables, other	0.48	0.48	0.48
POTATOES AND FRUITS			
Potatoes, canned	0.58	0.29	0.193
Potatoes, other	1.67	1.67	1.67
Other vegetables, canned	6.58	3.29	2.193
Other vegetables, other	3.69	3.69	3.69
Other vegetable juice, canned	0.15	0.075	0.05
Other fruits, canned	6.79	3.395	2.263
Other fruits, other	2.92	2.92	2.92
Other fruit juice, canned	0.61	0.305	0.203
Other fruit juice, other	0.03	0.03	0.03
TOTAL	107.475	94.085	89.561

TABLE A-3. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES AND FEMALES 3-5 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, µg	Lead Intake, µg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	7.91	7.91	7.91
Milk, processed, canned	4.16	2.08	1.387
Milk, processed, other	6.84	6.84	6.84
Cream	2.11	2.11	2.11
Cheese and mixtures	0.48	0.48	0.48
Eggs	4.0	4.0	4.0
MEATS			
Beef, canned	0.28	0.14	0.093
Beef, other	6.73	6.73	6.73
Pork, canned	0.22	0.11	0.073
Pork, other	5.75	5.75	5.75
Poultry	2.03	2.03	2.03
Fish, canned	0.58	0.29	0.193
Fish, other	1.93	1.93	1.93
Other and mixtures, canned	1.13	0.565	0.377
Other and mixtures, other	5.74	5.74	5.74
NUTS AND OILS			
Legumes and nuts	7.28	7.28	7.28
Fats and oils	0.24	0.24	0.24
BREADS			
Bread	5.29	5.29	5.29
Other baked goods	4.3	4.3	4.3
Cereals, pastes	8.88	8.88	8.88
SUGAR PRODUCTS			
Sugar	0.37	0.37	0.37
Candy, jam, etc.	2.17	2.17	2.17
BEVERAGES			
Tea	6.6	6.6	6.6
Coffee	0.35	0.35	0.35
Cola, canned	0.52	0.26	0.173
Cola, other	5.06	5.06	5.06
Fruit drinks, canned	0.26	0.13	0.087
Fruit drinks, other	0.57	0.57	0.57
Other soft drinks, canned	0.03	0.015	0.01
Other soft drinks, other	0.46	0.46	0.46
Beer, canned	0.0	0.0	0.0
Beer, other	0.0	0.0	0.0
Wine	0.0	0.0	0.0
Other alcoholic drinks	0.0	0.0	0.0
TOMATOES AND VEGETABLES			
Tomatoes, canned	2.09	1.045	0.697
Tomatoes, other	0.58	0.58	0.58
Tomato juice, canned	0.94	0.47	0.313
Citrus, canned	0.05	0.025	0.017
Citrus, other	1.28	1.28	1.28
Citrus juice, canned	1.45	0.725	0.483
Citrus juice, other	0.21	0.21	0.21
Dark green vegetables, canned	0.20	0.10	0.067
Dark green vegetables, other	0.13	0.13	0.13
Deep yellow vegetables, canned	0.29	0.145	0.0967
Deep yellow vegetables, other	0.48	0.48	0.48
POTATOES AND FRUITS			
Potatoes, canned	0.073	0.0365	0.0243
Potatoes, other	2.11	2.11	2.11
Other vegetables, canned	6.58	3.29	2.193
Other vegetables, other	3.69	3.69	3.69
Other vegetable juice, canned	0.15	0.075	0.05
Other fruits, canned	6.86	3.43	2.287
Other fruits, other	2.95	2.95	2.95
Other fruit juice, canned	0.61	0.305	0.203
Other fruit juice, other	0.03	0.03	0.03
TOTAL	123.023	109.786	105.354

TABLE A-4. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES AND FEMALES 6-8 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, µg	Lead Intake, µg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	8.36	8.36	8.36
Milk, processed, canned	4.40	2.20	1.467
Milk, processed, other	7.20	7.20	7.20
Cream	2.72	2.72	2.72
Cheese and mixtures	0.60	0.60	0.60
Eggs	4.002	4.002	4.002
MEATS			
Beef, canned	0.315	0.1575	0.105
Beef, other	7.52	7.52	7.52
Pork, canned	0.24	0.12	0.08
Pork, other	6.32	6.32	6.32
Poultry	3.175	3.175	3.175
Fish, canned	0.81	0.405	0.27
Fish, other	2.69	2.69	2.69
Other and mixtures, canned	1.50	0.75	0.5
Other and mixtures, other	7.25	7.25	7.25
NUTS AND OILS			
Legumes and nuts	9.36	9.36	9.36
Fats and oils	0.29	0.29	0.29
BREADS			
Bread	6.47	6.47	6.47
Other baked goods	5.40	5.40	5.40
Cereals, pastes	8.99	8.99	8.99
SUGAR PRODUCTS			
Sugar	0.43	0.43	0.43
Candy, jam, etc.	2.31	2.31	2.31
BEVERAGES			
Tea	7.2	7.2	7.2
Coffee	0.28	0.28	0.28
Cola, canned	0.68	0.34	0.227
Cola, other	6.56	6.56	6.56
Fruit drinks, canned	0.34	0.17	0.113
Fruit drinks, other	0.74	0.74	0.74
Other soft drinks, canned	0.03	0.015	0.01
Other soft drinks, other	0.60	0.60	0.60
Beer, canned	0.0	0.0	0.0
Beer, other	0.0	0.0	0.0
Wine	0.0	0.0	0.0
Other alcoholic drinks	0.0	0.0	0.0
TOMATOES AND VEGETABLES			
Tomatoes, canned	3.21	1.605	1.07
Tomatoes, other	0.89	0.89	0.89
Tomato juice, canned	1.45	0.725	0.483
Citrus, canned	0.05	0.025	0.0167
Citrus, other	1.31	1.31	1.31
Citrus juice, canned	1.47	0.735	0.49
Citrus juice, other	0.22	0.22	0.22
Dark green vegetables, canned	0.34	0.17	0.113
Dark green vegetables, other	0.212	0.212	0.212
Deep yellow vegetables, canned	0.34	0.17	0.113
Deep yellow vegetables, other	0.56	0.56	0.56
POTATOES AND FRUITS			
Potatoes, canned	0.09	0.045	0.03
Potatoes, other	2.61	2.61	2.61
Other vegetables, canned	7.81	3.905	2.603
Other vegetables, other	4.39	4.39	4.39
Other vegetable juice, canned	0.18	0.09	0.06
Other fruits, canned	7.67	3.835	2.557
Other fruits, other	3.30	3.30	3.30
Other fruit juice, canned	0.69	0.345	0.23
Other fruit juice, other	0.03	0.03	0.03
TOTAL	143.604	127.796	122.202

TABLE A-5. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES 15-17 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, ug	Lead Intake, ug/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	9.34	9.34	9.34
Milk, processed, canned	4.913	2.4565	1.638
Milk, processed, other	8.072	8.072	8.072
Cream	3.332	3.332	3.332
Cheese and mixtures	1.320	1.320	1.320
Eggs	7.308	7.308	7.308
MEATS			
Beef, canned	0.623	0.3115	0.2077
Beef, other	14.836	14.836	14.836
Pork, canned	0.447	0.2235	0.149
Pork, other	11.784	11.784	11.784
Poultry	3.429	3.429	3.429
Fish, canned	1.155	0.5775	0.385
Fish, other	3.845	3.845	3.845
Other and mixtures, canned	2.551	1.2755	0.8503
Other and mixtures, other	12.452	12.452	12.452
NUTS AND OILS			
Legumes and nuts	12.22	12.22	12.22
Fats and oils	0.507	0.507	0.507
BREADS			
Bread	11.34	11.34	11.34
Other baked goods	8.5	8.5	8.5
Cereals, pastes	9.951	9.951	9.951
SUGAR PRODUCTS			
Sugar	0.403	0.403	0.403
Candy, jam, etc.	3.22	3.22	3.22
BEVERAGES			
Tea	18.20	18.20	18.20
Coffee	4.13	4.13	4.13
Cola, canned	1.337	0.6685	0.4457
Cola, other	12.991	12.991	12.991
Fruit drinks, canned	0.666	0.333	0.222
Fruit drinks, other	1.451	1.451	1.451
Other soft drinks, canned	0.065	0.0325	0.0217
Other soft drinks, other	1.189	1.189	1.189
Beer, canned	0.0	0.0	0.0
Beer, other	0.0	0.0	0.0
Wine	0.0	0.0	0.0
Other alcoholic drinks	0.0	0.0	0.0
TOMATOES AND VEGETABLES			
Tomatoes, canned	4.658	2.329	1.553
Tomatoes, other	1.297	1.297	1.297
Tomato juice, canned	2.106	1.053	0.702
Citrus, canned	0.065	0.0325	0.0217
Citrus, other	1.780	1.780	1.780
Citrus juice, canned	2.010	1.005	0.67
Citrus juice, other	0.298	0.298	0.298
Dark green vegetables, canned	0.405	0.2025	0.135
Dark green vegetables, other	0.254	0.254	0.254
Deep yellow vegetables, canned	0.427	0.2135	0.1423
Deep yellow vegetables, other	0.723	0.723	0.723
POTATOES AND FRUITS			
Potatoes, canned	0.155	0.0775	0.0517
Potatoes, other	4.473	4.473	4.473
Other vegetables, canned	12.754	6.377	4.2513
Other vegetables, other	7.154	7.154	7.154
Other vegetable juice, canned	0.299	0.1495	0.0997
Other fruits, canned	8.013	4.0065	2.671
Other fruits, other	3.444	3.444	3.444
Other fruit juice, canned	0.716	0.358	0.2387
Other fruit juice, other	0.031	0.031	0.031
TOTAL	222.639	200.95	193.73

TABLE A-6. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: FEMALES 15-17 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, μg	Lead Intake, μg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	5.95	5.95	5.95
Milk, processed, canned	3.13	1.565	1.04
Milk, processed, other	5.14	5.14	5.14
Cream	2.312	2.312	2.312
Cheese and mixtures	0.96	0.96	0.96
Eggs	4.35	4.35	4.35
MEATS			
Beef, canned	0.48	0.24	0.16
Beef, other	11.47	11.47	11.47
Pork, canned	0.30	0.15	0.10
Pork, other	8.05	8.05	8.05
Poultry	2.29	2.29	2.29
Fish, canned	1.04	0.52	0.347
Fish, other	3.46	3.46	3.46
Other and mixtures, canned	1.84	0.92	0.613
Other and mixtures, other	9.19	9.19	9.19
NUTS AND OILS			
Legumes and nuts	7.54	7.54	7.54
Fats and oils	0.286	0.286	0.286
BREADS			
Bread	6.64	6.64	6.64
Other baked goods	5.90	5.90	5.90
Cereals, pastes	6.96	6.96	6.96
SUGAR PRODUCTS			
Sugar	0.31	0.31	0.31
Candy, jam, etc.	2.17	2.17	2.17
BEVERAGES			
Tea	15.2	15.2	15.2
Coffee	4.55	4.55	4.55
Cola, canned	1.13	0.565	0.377
Cola, other	10.94	10.94	10.94
Fruit drinks, canned	0.56	0.028	0.187
Fruit drinks, other	1.22	1.22	1.22
Other soft drinks, canned	0.055	0.61	0.018
Other soft drinks, other	1.001	1.001	1.001
Beer, canned	0.0	0.0	0.0
Beer, other	0.0	0.0	0.0
Wine	0.0	0.0	0.0
Other alcoholic drinks	0.0	0.0	0.0
TOMATOES AND VEGETABLES			
Tomatoes, canned	4.01	2.005	1.337
Tomatoes, other	1.12	1.12	1.12
Tomato juice, canned	1.82	0.91	0.607
Citrus, canned	0.053	0.0265	0.018
Citrus, other	1.45	1.45	1.45
Citrus juice, canned	1.53	0.815	0.543
Citrus juice, other	0.24	0.24	0.24
Dark green vegetables, canned	0.54	0.27	0.18
Dark green vegetables, other	0.34	0.34	0.34
Deep yellow vegetables, canned	0.29	0.145	0.097
Deep yellow vegetables, other	0.48	0.48	0.48
POTATOES AND FRUITS			
Potatoes, canned	0.85	0.425	0.283
Potatoes, other	2.46	2.46	2.46
Other vegetables, canned	10.15	5.075	3.383
Other vegetables, other	6.24	6.24	6.24
Other vegetable juice, canned	0.24	0.12	0.08
Other fruits, canned	7.67	3.835	2.56
Other fruits, other	3.30	3.30	3.30
Other fruit juice, canned	0.69	0.345	0.23
Other fruit juice, other	0.03	0.03	0.03
TOTAL	168.027	150.631	143.709

TABLE A-7. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES 20-34 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, µg	Lead Intake, µg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	4.94	4.94	4.94
Milk, processed, canned	2.60	1.30	0.867
Milk, processed, other	4.27	4.27	4.27
Cream	2.24	2.24	2.24
Cheese and mixtures	1.44	1.44	1.44
Eggs	9.57	9.57	9.57
MEATS			
Beef, canned	0.91	0.455	0.303
Beef, other	21.76	21.76	21.76
Pork, canned	0.54	0.27	0.18
Pork, other	14.08	14.08	14.08
Poultry	4.06	4.06	4.06
Fish, canned	1.62	0.81	0.54
Fish, other	5.39	5.39	5.39
Other and mixtures, canned	2.97	1.485	0.99
Other and mixtures, other	14.73	14.73	14.73
NUTS AND OILS			
Legumes and nuts	3.38	3.38	3.38
Fats and oils	0.20	0.2	0.2
BREADS			
Bread	10.08	10.08	10.08
Other baked goods	7.70	7.70	7.70
Cereals, pastes	8.99	8.99	8.99
SUGAR PRODUCTS			
Sugar	0.53	0.53	0.53
Candy, jam, etc.	1.89	1.89	1.89
BEVERAGES			
Tea	29.60	29.60	29.60
Coffee	29.54	29.54	29.54
Cola, canned	1.074	0.537	0.358
Cola, other	10.44	10.44	10.44
Fruit drinks, canned	0.53	0.265	0.177
Fruit drinks, other	1.17	1.17	1.17
Other soft drinks, canned	0.05	0.025	0.017
Other soft drinks, other	0.96	0.96	0.96
Beer, canned	1.05	0.525	0.35
Beer, other	11.22	11.22	11.22
Wine	2.569	2.569	2.569
Other alcoholic drinks	0.517	0.517	0.517
TOMATOES AND VEGETABLES			
Tomatoes, canned	6.10	3.05	2.033
Tomatoes, other	1.70	1.70	1.70
Tomato juice, canned	2.76	1.38	0.92
Citrus, canned	0.06	0.03	0.02
Citrus, other	1.57	1.57	1.57
Citrus juice, canned	1.77	0.885	0.59
Citrus juice, other	0.26	0.26	0.26
Dark green vegetables, canned	5.76	2.88	1.92
Dark green vegetables, other	0.30	0.30	0.30
Deep yellow vegetables, canned	0.47	0.235	0.157
Deep yellow vegetables, other	0.80	0.80	0.80
POTATOES AND FRUITS			
Potatoes, canned	0.16	0.08	0.053
Potatoes, other	4.72	4.72	4.72
Other vegetables, canned	15.50	7.75	5.167
Other vegetables, other	8.70	8.70	8.70
Other vegetable juice, canned	0.36	0.18	0.12
Other fruits, canned	6.52	3.26	2.173
Other fruits, other	2.80	2.80	2.80
Other fruit juice, canned	0.58	0.19	0.19
Other fruit juice, other	0.02	0.02	0.02
TOTAL .	273.52	247.73	239.26

TABLE A-8. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: FEMALES 20-34 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, μg	Lead Intake, μg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	3.17	3.17	3.17
Milk, processed, canned	1.67	0.835	0.557
Milk, processed, other	2.74	2.74	2.74
Cream	2.04	2.04	2.04
Cheese and mixtures	1.32	1.32	1.32
Eggs	4.70	4.70	4.70
MEATS			
Beef, canned	0.53	0.265	0.177
Beef, other	12.66	12.66	12.66
Pork, canned	0.293	0.1465	0.098
Pork, other	7.76	7.76	7.76
Poultry	2.667	2.667	2.667
Fish, canned	1.04	0.52	0.347
Fish, other	3.46	3.46	3.46
Other and mixtures, canned	1.80	0.90	0.60
Other and mixtures, other	9.22	9.22	9.22
NUTS AND OILS			
Legumes and nuts	6.24	[illegible]	6.24
Fats and oils	0.30	0.30	0.30
BREADS			
Bread	5.96	5.96	5.96
Other baked goods	5.20	5.20	5.20
Cereals, pastes	8.03	8.03	8.03
SUGAR PRODUCTS			
Sugar	0.40	0.40	0.40
Candy, jam, etc.	1.54	1.54	1.54
BEVERAGES			
Tea	23.80	23.80	23.80
Coffee	28.0	28.0	28.0
Cola, canned	0.59	0.445	0.297
Cola, other	8.71	8.71	8.71
Fruit drinks, canned	0.45	0.225	0.15
Fruit drinks, other	0.97	0.97	0.97
Other soft drinks, canned	0.04	0.02	0.013
Other soft drinks, other	0.80	0.80	0.80
Beer, canned	0.22	0.11	0.073
Beer, other	2.39	2.39	2.39
Wine	0.55	0.55	0.55
Other alcoholic drinks	0.11	0.11	0.11
TOMATOES AND VEGETABLES			
Tomatoes, canned	5.13	2.565	1.71
Tomatoes, other	1.432	1.432	1.432
Tomato juice, canned	2.33	1.165	0.777
Citrus, canned	0.05	0.025	0.017
Citrus, other	1.43	1.43	1.43
Citrus juice, canned	1.47	0.735	0.49
Citrus juice, other	0.22	0.22	0.22
Dark green vegetables, canned	0.54	0.27	0.18
Dark green vegetables, other	0.34	0.34	0.34
Deep yellow vegetables, canned	0.33	0.165	0.11
Deep yellow vegetables, other	0.56	0.56	0.56
POTATOES AND FRUITS			
Potatoes, canned	0.11	0.055	0.037
Potatoes, other	2.75	2.75	2.75
Other vegetables, canned	10.99	5.495	3.663
Other vegetables, other	6.16	6.16	6.16
Other vegetable juice, canned	0.24	0.12	0.08
Other fruits, canned	4.31	2.155	1.437
Other fruits, other	1.86	1.86	1.86
Other fruit juice, canned	0.42	0.21	0.14
Other fruit juice, other	0.02	0.02	0.02
TOTAL	190.36	173.935	168.463

TABLE A-9. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: MALES 55-64 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, µg	Lead Intake, µg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	3.15	3.15	3.15
Milk, processed, canned	1.66	0.83	0.533
Milk, processed, other	2.73	2.73	2.73
Cream	2.65	2.65	2.65
Cheese and mixtures	1.80	1.80	1.80
Eggs	8.87	8.87	8.87
MEATS			
Beef, canned	0.67	0.335	0.223
Beef, other	16.02	16.02	16.02
Pork, canned	0.50	0.250	0.167
Pork, other	13.08	13.08	13.08
Poultry	3.56	3.56	3.56
Fish, canned	2.08	1.04	0.693
Fish, other	6.92	6.92	6.92
Other and mixtures, canned	1.91	0.935	0.637
Other and mixtures, other	10.92	10.92	10.92
NUTS AND OILS			
Legumes and nuts	6.50	6.50	6.50
Fats and oils	0.46	0.46	0.46
BREADS			
Bread	8.99	8.99	8.99
Other baked goods	7.50	7.50	7.50
Cereals, pastes	7.81	7.81	7.81
SUGAR PRODUCTS			
Sugar	0.53	0.53	0.53
Candy, jam, etc.	2.10	2.10	2.10
BEVERAGES			
Tea	20.0	20.0	20.0
Coffee	39.06	39.06	39.06
Cola, canned	0.35	0.175	0.117
Cola, other	3.42	3.42	3.42
Fruit drinks, canned	0.18	0.09	0.06
Fruit drinks, other	0.38	0.38	0.38
Other soft drinks, canned	0.02	0.01	0.007
Other soft drinks, other	0.31	0.31	0.31
Beer, canned	0.70	0.35	0.233
Beer, other	7.51	7.51	7.51
Wine	1.72	1.72	1.72
Other alcoholic drinks	0.35	0.35	0.35
TOMATOES AND VEGETABLES			
Tomatoes, canned	4.81	2.405	1.603
Tomatoes, other	1.34	1.34	1.34
Tomato juice, canned	2.18	1.09	0.727
Citrus, canned	0.06	0.03	0.02
Citrus, other	1.57	1.57	1.57
Citrus juice, canned	1.77	0.885	0.59
Citrus juice, other	0.26	0.26	0.26
Dark green vegetables, canned	0.54	0.27	0.18
Dark green vegetables, other	0.34	0.34	0.34
Deep yellow vegetables, canned	0.62	0.31	0.207
Deep yellow vegetables, other	1.04	1.04	1.04
POTATOES AND FRUITS			
Potatoes, canned	0.13	0.065	0.043
Potatoes, other	3.74	3.74	3.74
Other vegetables, canned	13.72	6.86	4.573
Other vegetables, other	7.70	7.70	7.70
Other vegetable juice, canned	0.32	0.16	0.107
Other fruits, canned	7.54	3.77	2.51
Other fruits, other	3.24	3.24	3.24
Other fruit juice, canned	0.67	0.335	0.223
Other fruit juice, other	0.03	0.03	0.03
TOTAL	236.03	216.12	209.06

TABLE A-10. DAILY INTAKE OF LEAD FROM VARIOUS FOODS AND THE EFFECT OF REDUCING LEAD CONTENT OF CANNED FOODS: FEMALES 55-64 YEARS OLD.

Food Class and Substance	Normal Daily Lead Intake, µg	Lead Intake, µg/day, with Lead In Canned Food Reduced	
		Reduced 1/2	Reduced 2/3
MILK			
Milk, fresh	2.35	2.35	2.35
Milk, processed, canned	1.23	0.615	0.41
Milk, processed, other	2.03	2.03	2.03
Cream	2.38	2.38	2.38
Cheese and mixtures	1.68	1.68	1.68
Eggs	5.74	5.74	5.74
MEATS			
Beef, canned	0.44	0.22	0.147
Beef, other	10.68	10.68	10.68
Pork, canned	0.27	0.135	0.09
Pork, other	7.04	7.04	7.04
Poultry	3.18	3.18	3.18
Fish, canned	1.04	0.52	0.347
Fish, other	3.46	3.46	3.46
Other and mixtures, canned	1.65	0.825	0.55
Other and mixtures, other	8.73	8.73	8.73
NUTS AND OILS			
Legumes and nuts	4.42	4.42	4.42
Fats and oils	0.31	0.31	0.31
BREADS			
Bread	5.63	5.63	5.63
Other baked goods	5.1	5.1	5.1
Cereals, pastes	5.56	5.56	5.56
SUGAR PRODUCTS			
Sugar	0.28	0.28	0.28
Candy, jam, etc.	1.54	1.54	1.54
BEVERAGES			
Tea	21.80	21.80	21.80
Coffee	36.54	36.54	36.54
Cola, canned	0.30	0.15	0.1
Cola, other	2.96	2.96	2.96
Fruit drinks, canned	0.15	0.075	0.05
Fruit drinks, other	0.33	0.33	0.33
Other soft drinks, canned	0.01	0.005	0.003
Other soft drinks, other	0.27	0.27	0.27
Beer, canned	0.12	0.06	0.04
Beer, other	1.24	1.24	1.24
Wine	0.28	0.28	0.28
Other alcoholic drinks	0.06	0.06	0.06
TOMATOES AND VEGETABLES			
Tomatoes, canned	4.98	2.495	1.66
Tomatoes, other	1.39	1.39	1.39
Tomato juice, canned	2.25	1.125	0.75
Citrus, canned	0.07	0.035	0.023
Citrus, other	1.90	1.80	1.80
Citrus juice, canned	2.04	1.02	0.68
Citrus juice, other	0.30	0.30	0.30
Dark green vegetables, canned	0.47	0.235	0.157
Dark green vegetables, other	0.30	0.30	0.30
Deep yellow vegetables, canned	0.48	0.24	0.16
Deep yellow vegetables, other	0.80	0.80	0.80
POTATOES AND FRUITS			
Potatoes, canned	0.08	0.04	0.027
Potatoes, other	2.36	2.36	2.36
Other vegetables, canned	10.97	5.485	3.657
Other vegetables, other	6.15	6.15	6.15
Other vegetable juice, canned	0.26	0.13	0.087
Other fruits, canned	6.25	3.125	2.083
Other fruits, other	2.69	2.69	2.69
Other fruit juice, canned	0.56	0.28	0.187
Other fruit juice, other	0.02	0.02	0.02
TOTAL	183.02	166.215	154.458